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AUGUST, 1928

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CONTENTS OF PREVIOUS NUMBER

JULY, 1928. NUMBER 1

Purulent Meningitis: Contribution to the Clinical Picture. E. Schmiegelow, Copenhagen.

Contraction of the Visual Fields in Cases of Progressive Deafness: Report of Cases and a Comparison with Cases of Deafness of Other Types. Mark J. Gottlieb, M.D., New York.

Tracheobronchial Tree in Children, with Reference to Blood Aspirated During Tonsillectomy and Adenoidectomy Under Ether Anesthesia. Harris H. Vail, M.D., Cincinnati.

Ligation of the External Carotid Artery for Persistent Nasal Hemorrhage. Benjamin H. Abrahams, M.D., Brooklyn.

Radical Operation on the Mastoid: End-Results in One Hundred Unselected Cases. Leon E. White, Jr., M.D., Boston.

An Operation for the Relief of Abductor Paralysis of the Larynx. John Edmund MacKenty, M.D., New York.

Irrigations with Aqueous Solution: Their Effect on the Membranes of the Upper Respiratory Tract of the Rabbit. W. Berkeley Stark, M.B. (Tor.), Rochester, Minn.

The Histology and Pathology of the Articulation of the Auditory Ossicles. Joseph G. Druss, M.D., New York.

Congenital Esophageal Stenosis. Clyde A. Heatly, M.D., Rochester, N. Y.

A Suggestion Regarding the Treatment of Conditions Ordinarily Included Under Chronic Catarrhal Otitis Media. Louis K. Pitman, New York.

Clinical Notes:
- An Operative Procedure Suggested for Sinus Thrombosis. Jacob L. Maybaum, M.D., New York.
- A New Rhinoplastic Instrument. Joseph Safian, M.D., New York.
- The Degree of Malignancy of Carcinoma of the Esophagus. Albert C. Broders, M.D., and Porter P. Vinson, M.D., Rochester, Minn.

Progress in Otolaryngology. A Summary of the Bibliographic Material Available in the Field of Otolaryngology. The Paranasal Cavities: A Review of the Literature of 1927. D. Campbell Smyth, M.D., Boston.

News and Comment:
- American Board of Otolaryngology.

Correspondence:
- Treatment of Vincent's Angina. Joseph H. Abraham, M.D., New York.

Abstracts from Current Literature.

Society Transactions:
- Chicago Laryngological and Otological Society.
- College of Physicians of Philadelphia, Section on Otology and Laryngology.

Book Reviews.

Directory of Ophthalmological and Otolaryngological Societies.

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ARCHIVES OF OTOLARYNGOLOGY

VOLUME 8 AUGUST, 1928 NUMBER 2

THE TONSIL PROBLEM

THE RESEARCH WORK OF THE VIENNESE CLINIC *

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At a time when so much is being spoken and written about operative technicalities relating to the tonsils, it may be permissible to discuss also the problems of physiology and pathogenesis connected with the tonsils and the results of the investigations of the last years on this subject.

The newer investigations in which the Viennese Clinic, under the direction of its chief, Professor Hajek, has taken prominent part originated in the fundamental research work of Schlemmer. It deals with one of the chief problems of the entire tonsil question, namely, with the lymph streams to, in and from the tonsils.

To all laryngologists, the occurrence of tonsillitis after endonasal operations—even the slightest ones—is well known. This clinical observation has led to the widespread opinion that the lymph stream courses from the nose to the tonsils, and thus that the latter are the regional lymph glands of the nasal mucous membrane, or are the most forward-projected lymph glands of the neck. In connection with this question, which must, for all subsequent investigations, be of decided importance, Schlemmer [1] made histologic experimental and clinical studies. The conclusions drawn from his histologic studies are: The lymphoid tissue of the pharynx is not interpolated into the course of the lymph vessels, but is situated peripherally. This tissue, therefore, can be only the origin and source of the efferent lymph vessels and never a passage way and filtration station, as is actually the case in ordinary lymph glands. The lymphoid substance is only an integral part of the pharyngeal mucous membrane and is never an individual organ; therefore, one should not speak of the function of the tonsils alone, but of the function of the whole pharyngeal mucous membrane, a part of which is the lymphoid tissue, and therefore also the tonsils.

In the experimental studies of Schlemmer (in which dyes were injected into the nasal and pharyngeal mucous membrane of the living and into the pharyngeal mucous membrane of the dead), the following observations were made: The injected substance appears in the efferent

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* This article was translated by Dr. Ben L. Bryant, Cincinnati.

1. Schlemmer: Anatomische experimentelle und klinische studien von tonsillar Problem, Monatschr. f. Ohren., 1921.

vessels and in the regional lymph glands of the neck, but never in the tonsils and lymphoid tissues, even though they are entirely surrounded by the dye (fig. 1). This means that neither the palatine tonsils nor the remaining lymph tissue of the pharynx possesses afferent vessels. Also, injections into the tonsil itself or behind the tonsillar capsule showed without exception that the dye was transported only to the regional glands of the neck and never toward the pharynx. With this was

Fig. 2.—Metastases in the known regional glands but not in the tonsils. *A* indicates carcinoma; *B*, epithelioma of the pharynx and *C*, tonsil.

presented an important argument against all those theories which assume the presence of a pharyngeally-directed lymph stream and a protective action of the tonsils in this sense.

Finally, these histologic and experimental results are definitely proved by clinical observations. Carcinomas in the region of the sinuses, turbinates, soft parts of the pharynx, pillars, uvula and other areas cause metastases in the known regional glands, but never in the tonsils (fig. 2).

Fig. 1.—Injected substance appearing around the tonsil but not in the tonsil and lymphoid tissue. The pale area in the middle is the tonsil.

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These results proved that a tonsillar disease coming from the nose by way of the lymph stream does not exist; that the three tonsils are not lymph glands, and that the tonsillar tissue is not different from the remaining lymphoid tissue, which is divided in a greater or lesser degree throughout the pharyngeal mucous membrane.

As the lymph current from the pharyngeal mucous membrane follows the general law of flowing centripetally to the regional glands and not in the opposite direction, the same holds true for the tonsils, since they are merely a part of this mucous membrane.

There is still one point to be mentioned: In the lingual and pharyngeal tonsils, the contiguous mucous glands open into the crypts. In the palatine tonsils, this hardly ever occurs. In agreement with this anatomic fact is the clinical observation that plugs occur rarely in the lingual and pharyngeal tonsils, probably because here they are washed out by the mucus.

EXPERIMENTS

The research work of Schlemmer dealt with anatomic and physiologic details. In the subequent investigations of the Viennese clinic, the problem was attacked from the bacteriologic standpoint. To determine whether streptococci have anything to do with the clinical picture of tonsillitis, I had injected into my cubital vein 20 cc. of the blood of a patient with tonsillitis (whose temperature at that time was 103.1 F.) and one day later rubbed into my own tonsils, after previous scarification, a smear from the diseased tonsils. Both experiments gave negative results, as I had expected. A reaction could not be seen. One observation, however, pointed a certain way for further investigations. Having examined the phagocytic power of my blood against the streptococci from the patient before and after this experiment, I found that it had definitely increased following the injection. There then followed a further series of examinations in this direction. The conclusions from these were:

1. Tonsillitis cannot be transferred by inoculation from tonsil to tonsil or by the tranfusion of blood from diseased to healthy persons.

2. The phagocytic power of the patient's blood against the streptococci cultivated from the patient's tonsils increases in direct ratio with the stage of healing, and the resistance of the organism against these streptococci increases likewise.

3. This increase in resistance of the organism is directed specifically only against the strain cultivated from the patient; against foreign strains the organism does not show an increase in the power of defense with progress of healing.

4. The streptococci in persons with tonsillitis are not accidental and secondary symptoms, but are in close relation to the disease and its course.

That the streptococci play a part was proved by these investigations, but the question remains unexplained as to how the inactive streptococci, normally harmless parasites on the tonsils, become such dangerous factors in tonsillitis. The next series of investigations was dedicated to this theme. They aimed at the solution of the question: Is it possible to find any factor which enables the bacteria to develop and exhibit their power? If this possibility exists, it can occur only in an early stage before the typical clinical appearances characterize the disease. To find such cases is extremely difficult, but laryngologists, nevertheless, occasionally see such cases in which tonsillitis develops after nasal operations. Among numerous fruitless trials, it was possible in four cases to carry on the examinations at a time when clinical evidence of tonsillitis was not present, and the disease did not manifest itself until during the observation. When the blood from these four patients was used as culture medium for the streptococci taken from the tonsils of the respective patients, these streptococci became much more virulent than when they were cultivated on normal blood or bouillon, and caused the death of mice in a fraction of the time required by the same streptococci cultured normally. The blood of these patients alone, injected for control purposes, was not infectious in any case. The combination of the blood of the patient alone, followed six hours later by an injection of bouillon culture of his streptococci, caused the death of the mice in a short time. The blood of a patient who became ill twice during the period of observation, once with tonsillitis and later with an abscess, showed the faculty of increase in virulence only during the early stages of the first disease.

The following conclusions are the results of these observations: Some unknown factor (whether designated by the name disposition or some other term is unimportant) activates the streptococci in tonsillitis. Perhaps the common cold may conceivably play the rôle by acting primarily as a disturber of the biologic equilibrium and enabling the secondary factor, i.e., the germ, to play its part. It seems certain that intranasal operations also may cause such a disturbance of the biologic equilibrium of the entire organism, and thus prepare the way for tonsillitis. And so from another phase of the question one finds the same result as Schlemmer did on the basis of his anatomic investigations, i.e., tonsillitis occurring after nasal operations is not a regional reaction. Neither the last mentioned factor alone nor the streptococci alone are sufficient to cause the disease; there must be a combination of the two.

What takes place in the tonsils themselves during tonsillitis has been unknown until now. A proper consideration of this question was always prevented by the fact that the removal of tonsils during an acute inflammation was always avoided in this clinic. Finally, however, the desired material was assembled by reason of the occasional necessity of removing acutely inflamed tonsils in a suffocating child in order to obviate the

need of a tracheotomy. Thus, Grossmann and I [2] were able to study the exact changes within the tonsils during the course of acute inflammation.

Before I describe the results, the following preliminary statement may be made: Hitherto it has been believed that lacunar tonsillitis is a superficial disease of the tonsils with a leukocytic exudate in the foreground which fills the crypts as plugs, and that in the lymphoid tissue itself, great changes are not found, at most, a swelling of the lymph follicles is seen. The lymph follicles, which characterize every lymphoid tissue, are at present in the foreground of scientific discussion of lymphoid tissue. It is not known what they are nor what function they

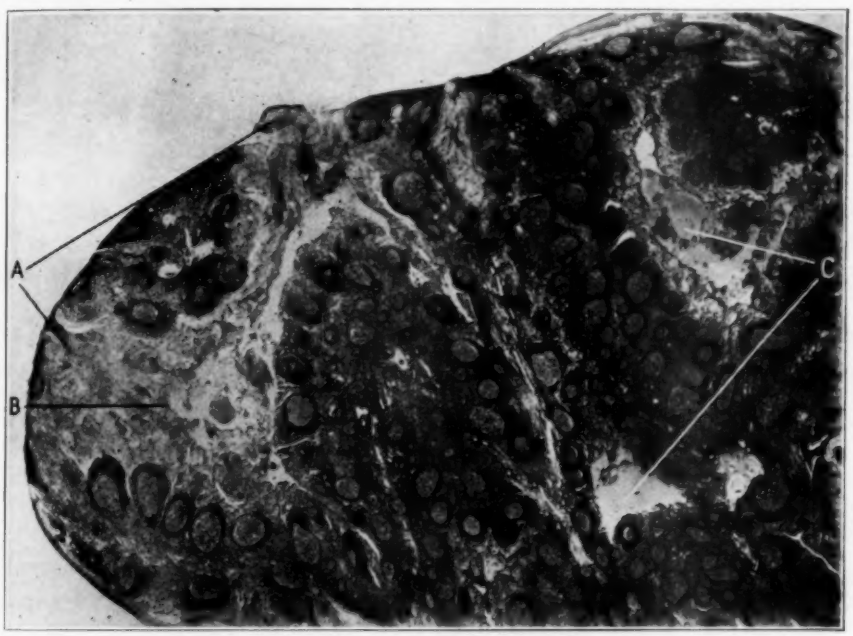

Fig. 3.—Extension of pathologic anatomic changes into a great part of the tonsillar parenchyma. *A* indicates plugs; *B*, destroyed parenchyma and *C*, crypts.

have. For many years the lighter inner zone, which is sharply bordered by a dark outer wall of lymphocytes, was considered a so-called germ center with the hypothetic function of producing lymphocytes. This inner zone contains a certain type of large cells—lymphocytes, never leukocytes. Even Flemming,[3] one of the earliest to describe these formations, found the so-called "stainable bodies" (tingible Körper) in the germ centers. He could not find an explanation for these and could not

2. Grossmann and Waldapfel: Lacunar Tonsillitis, Acta oto-laryng., 1926, vol. 10, part 1.

3. Flemming: Arch. f. mikr. Anat., 1885, vol. 24.

bring them into harmony with the theory of producing lymphocytes. Therefore, another explanation has been constantly sought recently. This will be discussed later in connection with the results of the experiments performed in this clinic.

Grossmann and I have found that in the diseased tonsils the pathologic anatomic changes not only are superficial, but extend into the depth and concern a great part of the tonsillar parenchyma (fig. 3). The phlegmonous changes are in the foreground. Deep destruction of the lymphoid tissue is found even in the first days of the disease. Purulent foci of destroyed tissue, varying in number and extent, occur. What appears clinically as a plug not only is a cast of a lacuna, but is as often a branch of such a purulent focus of the tonsillar parenchyma

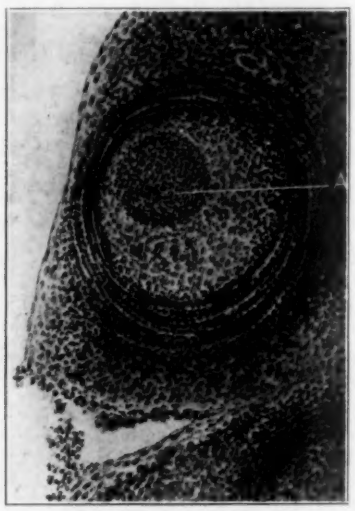

Fig. 4.—Aggregations of polymorphonuclear leukocytes and streptococci in the inner parts of the follicles. *A* indicates focus of leukocytes.

extending to the surface. Exudate, as well as destroyed tissue, always contains the same flora of bacteria: a pure culture of gram-positive diplococci and streptococci.

The most interesting changes of the parenchyma appear in the lymph follicles. In the earliest stages of the disease which I was able to study, aggregations of polymorphonuclear leukocytes and streptococci often existed in the inner parts of the follicles (fig. 4). These aggregations most often lie centrally, sometimes more excentrically; they seldom perforate the wall of lymphocytes and only rarely have a direct communication with the lacunae. In slightly more advanced stages, these foci no longer contain cocci, and soon thereafter they are not found any more.

The condition of the pharyngeal tonsil is also important. It may show the same changes as the palatine tonsils or, in spite of the most severe reactions in the latter, may remain intact.

CONCLUSIONS

Important conclusions from these observations are: Lacunar tonsillitis is not a superficial disease but is a deeply extending destructive disease of the tonsillar parenchyma. The pictures correspond to the phlegmonous stage of appendicitis described by Aschoff. Perforations

Fig. 5.—Perforations of the epithelium corresponding to Aschoff's primary infections. *A* indicates perforation of the epithelium (primary infection) and *B*, epithelium of the crypt.

in the epithelium corresponding to Aschoff's "primary infections" are also found (fig. 5). These pathologic anatomic changes also fit in much better with the severity of the clinical picture. Lacunar tonsillitis is not a simple catarrhal infection.

In regard to the name, the clinical name and the pathologic anatomic observations do not correspond entirely. Until now both designations, "lacunar" and "follicular" tonsillitis, have been used without a pathologic

anatomic basis. My observations admit both and show that both are incomplete, because at the climax of the disease, both lacunar and follicular changes are in the foreground. These follicular changes perhaps constitute the most important part of the research. Neither leukocytes nor cocci belong to the normal picture of the follicles. The presence of aggregations of this kind has never before been proved with certainty, not even in acute appendicitis, which, in Aschoff's own words, bears a striking resemblance to acute tonsillitis.

Up to the present time the prevailing opinion is that the infection passes from the surface into the depth after breaking through the epithelium of the lacunae. I do not wish to state what is not yet proved, but on observing such a picture as figure 4 presents, which also shows many foci confined to the centers of the lymph follicles, an unbiased

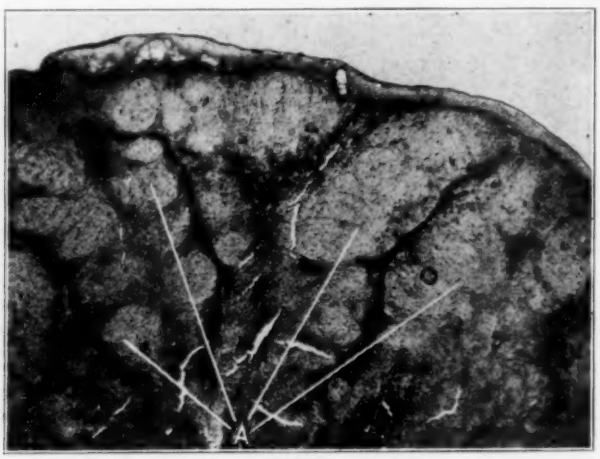

Fig. 6.—Normal tonsillar section under oxydase resection. *A* indicates follicles.

observer must admit that the infection may take the opposite course, an opinion which gains more and more ground clinically. Bacteriologic examinations of these sections prove the results obtained in my former research work, namely, that streptococci are not accidentally found in tonsillitis, but that they are a part of the picture of the disease.

Finally, the condition in the lymph follicles is of importance in another regard. I have mentioned that the belief that the lymph follicles are exclusively places of production of lymphocytes fails to correspond with different observations made within them, and recently, doubts of this singleness of function have increased. On the basis of his own observations and experiments, Hellmann [4] was the first to express a new

4. Hellmann: Studien über das lymphoide Gewebe, Beitr. z. path. Anat. u. z. allg. Pathol., vol. 68.

theory which sees in the lymph follicles "reaction centers" against invading toxins (bacteria and other toxic products). According to this theory, the "stainable bodies" could also be explained easily: They are an expression of degenerative processes caused by the toxic products, and represent nuclear fragments. To be sure, Hellmann admitted that if this theory would hold good, bacteria—or rests of them—should be found within these nuclear fragments; but he was not successful in finding them in spite of repeated examinations. "If this opinion could be firmly established through closer examination, it would give a strong support to the idea that lymphatic tissue acts as a protecting wall against bacterial and toxic products invading the organism. Thereby the anatomic basis for the function of the lymphatic tissue would be given."

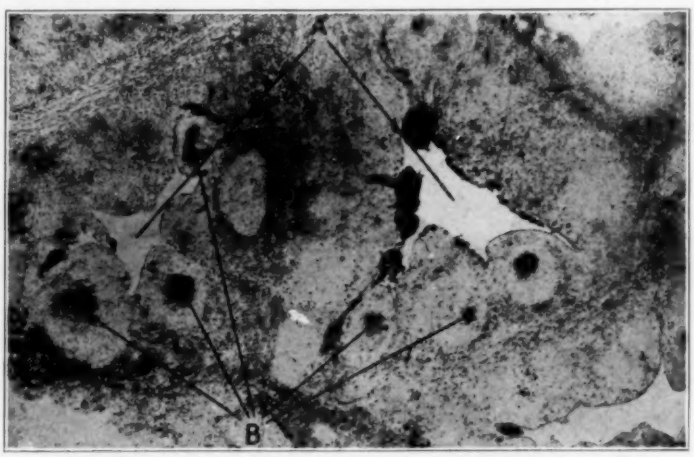

Fig. 7.—Section from area showing climax of the disease. *A* indicates crypts and *B*, foci of leukocytes in follicles.

In the research work in this clinic, we were successful in finding the bacteria supposed by Hellmann to be present in the lymph follicles, and were able to contribute to the solution of this question.

The rôle which the leukocytes played in this work caused me to dedicate an entire series of investigations [5] to them, and I have made a series of oxydase reactions from patients in different stages of tonsillitis. Figure 6 shows a normal tonsillar section under oxydase reaction; figure 7, a section from an area showing the climax of the disease. While in the former all follicles are free, dark foci of leukocytes are seen

5. Waldapfel: Etologie der Angina, Monatschr. f. Ohrenh., 1923, vol. 57; Neue Beitrage f. Anginaproblem, ibid., 1924, vol. 58; Weitere Untersuchungen über Veranderungen in den Lymphfollikeln, Ztschr. f. Hals- Nasen- u. Ohrenh., 1927, vol. 17.

in the latter. The oxydase reaction, which brings out all these foci, shows them to be much more numerous than could be accepted at the first examination. In the course of the next days, these foci disappear, and in the inner part of the lymph follicles more and more large cells are to be seen loaded with black granules—perhaps cells of the follicles which have taken up degenerated leukocytes. Figure 8 shows such a follicle which takes on a peculiar appearance because of these cells.

Fig. 8.—Peculiar appearance of follicle because of large cells loaded with black granules. *A* shows large cells loaded with black granules.

In another series of examinations, sections of tonsils examined a few days after the climax of the disease, did not show a less remarkable picture when examined for fatty degeneration. Figure 9 shows the same condition as figures 4 and 7.

One fact has been brought out by this work: Whatever one's opinion may be of the function of the lymph follicles and the germ centers, they are not exclusively, if at all, centers of production of lymphocytes.

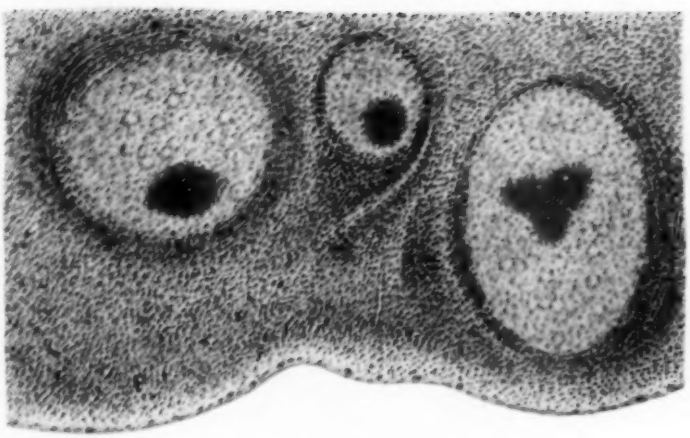

Fig. 9.—The same condition as is shown in figures 4 and 7. The orange areas in the center show foci of fatty degeneration.

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COMMENT

This short review of the work done in the Viennese clinic on the tonsil problem should serve to present a picture of the difficulties that beset one on every hand: how much is known and how much more is not known. Much remains to be solved. Only building stones have been presented here, but they are the foundation stones on which more can and will be built, and by further indefatigable effort, the edifice will finally be completed.

ASEPTIC CAVERNOUS SINUS THROMBOSIS

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Two years ago, I discussed lateral sinus thrombosis before the ophthalmologic and otolaryngologic section of the Oklahoma State Medical Association. The paper was presented without manuscript, and a brief was not prepared, so the discussion was not received for publication by the *Journal of the Oklahoma State Medical Association*. At that time, after several years of observation, I presented conclusions which were, I believe, not generally accepted; namely, (*a*) all lateral sinus thrombi are aseptic during the formative period; (*b*) lateral sinus thrombosis does not come as the result of infecting micro-organisms passing directly into the lateral sinus or from an infected embolus entering the sinus by way of the superior petrosal or some other communicating vessel.

I endeavored to show that a thrombus was the result, rather, of extension by contiguity of tissue. Following inflammatory processes within the temporal bone due to infection the membranous sinus becomes surrounded by inflammatory tissue and frequently lies in a pool of pus. As a result of this contiguous inflammation the vein becomes involved, causing the intima of the vessel to throw out a sticky exudate which picks up the red and white blood corpuscles and other blood contents, forming an aseptic thrombus. If the pus is not removed by prompt operation, the vessel walls soon soften; then the surrounding bacteria readily penetrate them, and the thrombus becomes infected, so that the patient presents a new syndrome, and the blood picture is that of bacteremia. If, at this time, operation is not performed the infected thrombus begins to break down, the patient develops a third distinct stage and the symptoms and blood picture are those of septicemia.

I urged the importance of recognizing the condition and performing an operation in these cases during the first stage, namely, that of the aseptic thrombus, when one may expect practically 100 per cent of recoveries. If one does not recognize this condition early but operates during the second stage, namely, that of bacteremia, only about 50 per cent of recoveries may be expected, whereas if the operation is delayed until the third stage, or that of septicemia, the mortality is practically 100 per cent. As an aid to diagnosis, blood cultures should be made daily in all cases of mastoiditis.

Those who have studied thrombi carefully in their gross appearance must have noted them occasionally during this aseptic stage. They are

cleancut, well molded structures, firm and adherent to the inner wall of the vessel, almost like an outgrowth from the intima. They are red, dark pink or pale pink dependent on the proportions of the white to the red blood corpuscles they contain. Microscopic examination does not reveal any bacteria in these thrombi during their formative stage.

Unfortunately, too few lateral sinus thrombi are seen at this stage because of the tendency to delay operation; or if one does perform a mastoidectomy, one so often fails to expose the lateral sinus, and hence the life of the patient may be seriously jeopardized if not wholly sacrificed.

As a result of the studies which led to the foregoing comment, I was able to make my deductions in the interesting case which I will report and which at the June, 1927, staff meeting at the University Hospital I called an aseptic cavernous sinus thrombosis. More than four months later, in the September, 1927, number of THE ARCHIVES OF OTOLARYNGOLOGY, Dr. M. C. Myerson [1] of New York presented a similar case which he called an aseptic cavernous sinus thrombosis. He stated that he was "not certain a case has ever before been recorded in which the process was aseptic apparently from its inception."

REPORT OF CASE

R. S., a colored school boy, aged 15, admitted to the University Hospital on May 3, 1927, was apparently perfectly well until one week before, when he began to have some pain with a rapid swelling and effusion of the left eye and orbit. He did not complain of any other symptoms.

The observations of the admitting physician, Dr. R. Brady, were "swelling of tissues about the left eye and lids, redness of conjunctiva and purulent discharge from the eye." It was the impression of the admitting physician that the condition was cellulitis of the left orbit.

The history and physical observations made by Dr. E. Veatch were entirely negative except, as related previously, for a rapid swelling and inflammation of the left eye which began seven days prior to admission into the hospital.

The patient was referred to the Ophthalmological Service, and Dr. E. S. Ferguson's consultation report on the case was as follows:

"Right eye: About normal in appearance, media all clear and fundus shows no pathology. There may be slight exophthalmos.

"Left eye: Marked exophthalmos with conjunctival edema. The appearance suggests cellulitis but there is a complete absence of pain, which would discount this opinion. Media clear, nerve head very indistinct with probably some exudate at its center presenting slight choked appearance. Unable to say what is causing disturbance without further study. May be cavernous but history hardly bears it out. Cellulitis from an infection should be more acute. Intracranial pressure of some sort must be considered. Advise close observation."

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1. Myerson, M. C.: Some Phases of Accessory Sinus Disease, Arch. Otolaryng. **6**:217 (Sept.) 1927.

Dr. L. M. Westfall, on service in the ophthalmologic department, called for a nose and throat consultation, with special reference to a possible infection from the accessory nasal sinuses.

Dr. J. C. Macdonald, of the rhinologic department, who was assisting me at this time in my service at the University Hospital, made the following observations on May 7, 1927:

"Right eye: Some exophthalmos, conjunctiva congested and edematous, movements of eyeball somewhat limited. Vision apparently good.

"Left eye: Marked exophthalmos, eyeball pushed forward and rotated downward, only slightly movable.

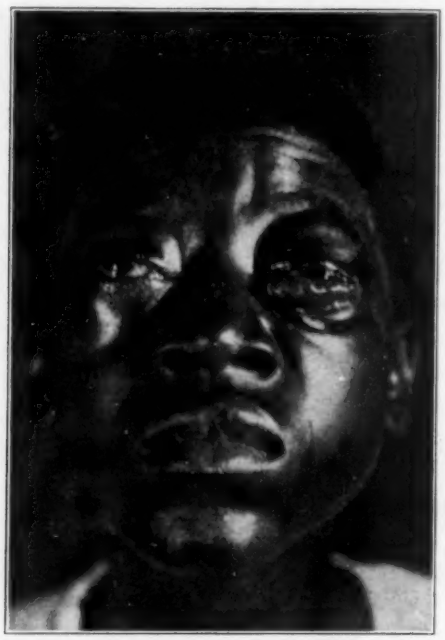

Fig. 1.—Condition of patient one week after entrance to the hospital. Exophthalmos in both eyes with marked chemosis of the conjunctiva of the left eye should be noted.

"The X-ray shows fluid in frontal sinus but as the sinus lights up well on transillumination, this is to be questioned.

"Impressions: Orbital cellulitis, bilateral. Dr. Macdonald asks an opportunity for further study."

On May 9, 1927, he made the following record: "Tissues of nose shrunken with adrenalin. . . . No pus is seen in either nasal cavity."

Dr. MacDonald kindly asked me to see the patient, which I did on May 11, 1927. At this time we made the following notes: "Condition and appearance of the patient remained as already described. This is probably a cavernous sinus thrombosis. Patient examined rather hurriedly. Extension from the nose has evidently been eliminated. It is probably a low grade mild type of

infection involving the cavernous sinus. We think the continued careful observation of the case important with as near as possible a positive elimination of the accessory nasal sinuses."

On June 8, 1927, we made the following notation regarding the case: "The condition of the fundi is not changed since first seen, which by the way, evidently had shown marked engorgement of both disks—except that the veins may be more swollen and the arteries, especially in the left eye, very indistinct. Both eyes still markedly bulging but the exophthalmos and chemosis is much more extreme in the left eye. This we contend is probably due to a low grade infection of the left sphenoidal sinus, causing an aseptic thrombus of the cavernous sinus. This may become septic at any time presenting serious complications or it may clear up by absorption."

Fig. 2.—Condition of patient two weeks after entrance to the hospital. The conjunctiva protruding below the margins of the closed lids of the left eye should be noted.

The outstanding features in this case and those on which our judgment in the diagnosis of aseptic cavernous sinus thrombosis was based, were: the history of a rapidly developing bilateral exophthalmos, more marked in this instance in the left eye, attended by little pain but marked redness of the conjunctiva, with engorgement and chemosis; evidence of intracranial pressure, as shown by a slow full pulse rate of from 52 to 56, and the enormously engorged veins in both fundi, indicating an obstruction to the venous circulation in both eyes; total absence of fever or other signs of sepsis, the red and white blood count remaining practically normal throughout, according to the laboratory reports.

The patients evidently tend to recover without surgical intervention. Dr. Myerson's patient recovered after seven months.

It is my opinion however, that if the intracranial pressure continues to be pronounced, as evidenced by the slow, heavy pulse rate, and the intense engorgement of the retinal veins, a subtemporal decompression should by all means be done to lessen the danger of blindness from intracranial pressure on the optic nerve. J. V. Paterson,[2] of the Royal Infirmary, Edinburgh, in a recent article on "Optic Neuritis," said, "I would support the view that cases of increased intracranial pressure with changes at the disk should be operated upon early so that the patients may have the best chance of retaining vision." A. J. Ballantyne, of the Western Infirmary of Glasgow, Scotland, and Henning Ronne,[3] of Copenhagen, also made the same claims in recent published articles.

Fig. 3.—Condition of patient eight months after entrance to the hospital. The normal appearance of both eyes should be noted.

As consultant, I recommended that a subtemporal decompression be performed in this case. The ophthalmologist in charge concurred in this opinion, and referred the case to the surgical department. Dr. LeRoy Long, who was on service, performed this operation on May 18, 1927. The patient had fully recovered after seven months, as the accompanying figures show.

X-ray and all other laboratory observations remained practically negative throughout the entire illness. The temperature remained nearly normal, whereas the pulse rate prior to the decompression operation was often below 55 per minute.

Dr. Myerson's patient recovered, apparently, without loss of sight, and was not operated on. The physician does not state, however,

2. Paterson, J. V.: Optic Neuritis, Eye, Ear, Nose & Throat Monthly **6**: 676 (Jan.) 1928.

3. Ronne, Henning: Nomenclature of Optic Neuritis, Eye, Ear, Nose & Throat Monthly **6**:679 (Jan.) 1928.

whether or not his patient showed any evidence of intracranial pressure. He does not tell us the character of the pulse rate nor the condition of the fundi. Several of the best authorities state that when the pulse rate remains about 55 or lower, owing to evident intracranial pressure, and the optic disk shows marked disturbance, a subtemporal decompression should always be done. I am inclined to believe that such an operation had much better be advised than to risk the possibility of partial or complete blindness.

How does one account for such a case as this? I believe that the etiology as based on my discussion of thrombosis two years ago will explain these conditions. This patient undoubtedly developed first a mild infection of the sphenoid, sufficient to cause an inflammation of the contiguous cavernous sinus, the intima of the cavernous sinus throwing out sufficient exudate to pick up the red and white blood cells and blood platelets to produce a thrombus. Fortunately, the sphenoidal sinus has a rather large opening for natural drainage, and, in case of infection, always tends to spontaneous cure. Resolution took place naturally, therefore, in the sphenoidal sinus, before the infection had broken down the membranous walls of the cavernous sinus to permit the bacteria to infect the thrombus. Such good fortune is impossible in lateral sinus thrombosis from purulent mastoiditis, but should be accomplished quite readily in cavernous sinus thrombosis through the natural drainage of the sphenoidal sinus.

From my observation of this case, I believe, however, that the safety of these patients could be greatly increased by the prompt administration of some intranasal treatment that would favor natural drainage of the accessory nasal sinuses, such as keeping the nasal mucous membrane shrunken with a 1 per cent solution of ephedrine or 1 : 1,000 epinephrine hydrochloride, followed by a gentle and persistent intranasal application of a hot alkaline douche.

The pictures presented with this report tell one more at a glance than pages of descriptive writing could possibly do, especially as to the appearance of a patient with cavernous sinus thrombosis.

309 Colcord Building.

FRONTAL-ETHMOSPHENOIDECTOMY

FURTHER EXPERIENCE WITH THIS OPERATION PERFORMED UNDER LOCAL ANESTHESIA; A NEW OSTEOPLASTIC FLAP *

EDWARD CECIL SEWALL, M.D.
SAN FRANCISCO

In 1926, I described before the American Laryngological Association an operation on which my co-workers and I had been at work for a number of years, and which at that time we had performed thirty times.[1] The two years since have given my associates and me more experience with this method, and I am grateful for the privilege of reviewing the work and profiting by the criticism made by the members of the association. My associate, Dr. Bacher, the house officer, Dr. Hunnicutt or I have operated on forty patients since the former report.

In the matter of diagnosis, we have made some progress through the microscopic examination of the discharge from the nose and also the antral contents. The cytologic study has been a help in establishing the fact of sinus infection and in localizing it. We do not consider that the operative indications have been materially changed. We advocate, as before, extreme conservatism in determining the necessity for operating on patients with sinus disease. The establishment of the diagnosis should be given whatever time and patience may be required. When the fact of sinus infection has been established, in doubtful cases we often watch the patient for months or years to estimate, if possible, the character and constancy of the infection and the significance it may have with respect to the patient's health. Many tire and do not return for this apparently fruitless observation. We feel that by this conservatism patients are allowed to carry with them a condition that frequently works constantly and insidiously in undermining their health, leading to arthritis, high blood pressure and deafness—to mention only a few of the well known results of chronic infection. Our judgment, however, in this matter is based on our distaste for operating on patients with low grade sinusitis when we are not certain that such resultant pathologic changes are imminent and on the probability that our operative methods may fall short of our ideal.

Before he is operated on the patient must be disturbed seriously enough by his sinus infection to warrant the belief that he will be distinctly improved by treatment. When operation is advised our experi-

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* Read before the American Laryngological Association, Washington, May, 1928.

1. Sewall, E. C.: External Operation on Ethmosphenoid Frontal Group of Sinuses Under Local Anesthesia, Arch. Otolaryng. **4**:377 (Nov.) 1926.

ence and results with the external method of operation lead us to advise this procedure with less hesitation as we realize its advantage over the intranasal method. We find also that our patients accept it with less reluctance, as while waiting they have sometimes talked with others who have been operated on.

Ligation of the vessels has proved consistently satisfactory. Believing that it would be dangerous to leave metal clips in place on account of the liability of aspiration, we developed the technic to ligate the vessels with catgut. This we will continue to use on the sphenopalatine artery where it crosses the body of the sphenoid, but there can be little if any objection to the use of the clips on the ethmoid arteries and none to their extramucosal application to the terminal branches of the internal maxillary artery at their entrance to the nose. The use of clips should save valuable time.

The field is practically bloodless, and the work progresses in a manner comparable only to that on the cadaver. Each cell is seen and thoroughly studied before it is opened. The thoroughness with which this study can be carried on is exemplified in distinguishing posterior ethmoid cells from the sphenoid. These large ethmoid cavities frequently simulate the sphenoid sinus, and careful study with the searcher and probe in a bloodless field is sometimes necessary to outline the sphenoid and find its opening.

Better anesthesia is attained by the blocking of the ethmoid nerves supplemented by the application of cocaine intranasally. The patient lies drowsily inattentive. The scopolamine and morphine are ideal in their action. Rarely, a high-strung patient will become a little upset and may even vomit. This is provided for by sterile towels under the gauze mouth drape; even then the patient scarcely ever remembers the occurrence.

Unusual location of diseased cells complicate our task. The supra-orbital cells extend outward over the orbit in the frontal bone. The supracanalicular extend in the lesser wing of the sphenoid over the optic canal, and sometimes even the lateral wall of the canal is hollowed. The sphenoid sinus frequently extends laterally into the greater wing or into the pterygoid process of the sphenoid bone. These extensions lie lateral to the plane capable of being reached by intranasal operation. The supra-orbital cells can usually not only be opened by the external technic, but removal of their orbital walls leads to their obliteration in almost all cases.

Recently, however, we have come to realize a formerly unrecognized stumbling block to progress in the eradication of ethmoid disease. This difficulty is encountered in the treatment of what may well be called the infra-orbital ethmoid cells. These cavities, often large, occupy lateral extensions in the ethmoid and palate bones and frequently lie over the

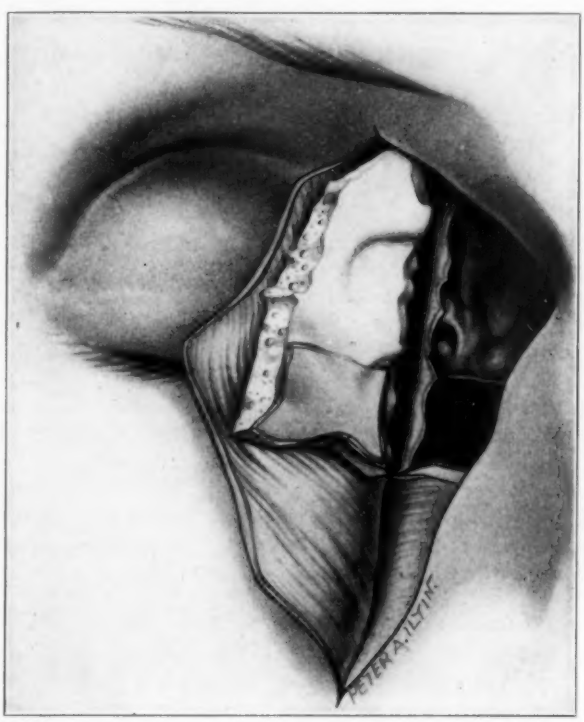
antrum and even behind the upper part of the posterior antral wall. We do not believe that they can be drained properly by any except the transantral route. This leads to the thought that the radical operation on the antrum and transantral opening of these cells should precede the frontal ethmosphenoid technic when pansinusitis requires operation on the antrum. We are at present working on this problem. The stereoscopic x-ray films will demonstrate the location of these cells as they do the supra-orbital extensions. When these classes of cells are wanting, intranasal operation may be satisfactory. It is doubtful, however, if in such apparently suitable cases one would have the temerity to break through some of the heavy cell walls that are plainly seen and easily removed when performing the external operation.

The deformity following the operation has been found to be practically negligible. Even in the three cases in which reoperation was required, the patients were not badly scarred. A few patients have rather ugly lines, and in some the retraction at the upper inner angle of the orbit is noticeable. In a number of cases, all marks of the operation defy detection except with trained scrutiny.

The most radical change that has been made in the original technic is the use of an osteoplastic flap at the site of the incision, as shown in the illustration. The reason for this is obvious to any one who has attempted the discouraging task of curing a patient with disease of the frontal sinus without using the deforming obliterative methods. The earlier writers, Kuhnt, Ogsten, Luc and others, attempted obliteration of the sinus. The result was unsightly, but in small sinuses must have been satisfactory from a curative standpoint. Killian was outstanding in his bold attack. The disfigurement following the retraction of the soft parts was extreme in some cases. It is possible that large frontal sinuses demand such obliteration to effect a cure. Another and more fundamental objection to Killian's technic is the fact that the drainage from the frontal sinus, attained immediately and so perfectly by the operation, is frequently diminished or entirely cut off by subsequent retraction of the soft parts following the window resection of the frontal process of the superior maxilla and frontal bone. If this obliteration of the frontonasal opening occurs before healing in the frontal sinus has taken place, abscess may form, requiring drainage.

When I wrote my former paper, I thought we had conquered this difficulty by the passing of sounds through the frontonasal opening. We have been successful in treating some patients by that means. In the case of one patient, reported at that time as requiring reoperation, we were able to establish a permanent opening into the frontal sinuses. In other cases, we have managed to open and keep open intranasally these sinuses after their closure had led to the formation of abscesses.

Many patients have recovered with little if any opening into the frontal sinuses. The inadequacy of the operation in this respect has led to much experimentation. In our endeavor to keep an opening patulous, we have added the principle of Lothrop to the technic in several cases. The space between the cribriform plate and the bony arch at the root of the nose is cut away according to his original and excellent idea, but by a different means. He does his work with the bur, working upward through the nose. We cut the partition between the two frontal sinuses



The osteoplastic flap is seen turned outward, broken away from the thin lamina papyracea. The nasal mucosa is shown attached to the bone. The anterior ethmoids are exposed to view. The retractor will be introduced between the orbital periosteum and the lamina and the parts drawn gently outward.

and work downward and upward with a chisel or a punch proceeding entirely through our window opening.

We recently had to reoperate on a patient because of acute abscess in the frontal sinus, which occurred many months after an apparently successful operation. We employed the Lothrop principle and secured primary union and relief from all symptoms. Our anxiety, however, is not entirely relieved, because shortly after the second operation, an

opening into the frontal sinus could not be found with the probe. We are convinced that sometimes an opening exists which takes care of the situation, but which cannot be found with the probe.

AN OSTEOPLASTIC FLAP

Ziegelmann, of our clinic staff, made the suggestion that an osteoplastic flap might be useful. I immediately worked out such a flap on the cadaver. Its preparation is not different from the technic for cutting the window in the bone, described in my former paper, except that the window of bone cut out is retained and replaced at the conclusion of the operation. My former description of this part of the operation is as follows:

> The window of bone to be removed is estimated, and with a sharp 8 mm. flat chisel, the lower limit of the bony window, usually a few millimeters below the upper end of the lacrimal fossa, is outlined. The chisel is worked through gradually until the nasal mucosa is reached. Care is taken not to cut through this nasal mucous membrane. The cut through the bone is made below first, while the upper parts are still intact. This lends firmness and assists the cutting. With a gouge, the thin bone at the inner upper angle of the orbit covering the frontal sinus is removed, and the frontal sinus is entered. If it is normal, it is not disturbed, though a probe is passed downward into the nose to outline the anterior edge of the bony window to be removed. The anterior edge of the bony incision is now outlined with the flat chisel. This joins the lower bony cut with the opening in the frontal sinus. After the bone has been cut through, the chisel is used as a lever and the bony window is pried gently upward. It is grasped in a small, rongeur forceps and is removed in one piece or when this fails, piece-meal. The external posterior edge of the bony window, which includes the attachment of the frontal process of the superior maxillary bone with the lamina papyracea and the lacrimal bone, is ignored, as it breaks easily.

Formerly, skin and periosteum were dissected off the bone before the window was cut. In the present method, skin, periosteum and nasal mucosa must be left strictly undisturbed on the bony flap. This makes the technic more difficult and has slowed the operation materially, but we hope that experience and practice will restore celerity. After the incision is made through the skin, the periosteum is cut and the skin retracted just enough at the top and bottom of the incision to allow for the making of the bony flap. After the bone has been penetrated with the chisel, the nasal mucous membrane lining is cut through with a knife, and the flap, covered by periosteum and skin and lined with mucosa, is pried outward with a chisel and the bone broken at the attachment of the frontal process of the maxilla with the thin bone covering the ethmoid. It breaks easily at that place.

The flap is retracted so that the periosteum of the orbit can be separated from the lamina papyracea and the retractor introduced as

heretofore described. From this point, the operation proceeds according to the technic formerly outlined.

We have operated on only two patients by this flap method, but we have repeatedly demonstrated its feasibility on the cadaver. It remains to be seen whether this will represent a real improvement. At present, these patients have excellent openings into the frontal sinuses, but that is not remarkable at this time.

The preservation of the nasal mucosa should prove to be an important refinement. It is hoped that it will decrease the tendency toward the formation of granulation tissue.

The most difficult part of the operation is the treatment of the sphenoids. It is hardest here to live up to the standards set by illustrations. I am convinced that the principle advocated is correct and attainable. Patients on whom we have employed the technic in proper detail have recovered. Others, when enough of the floor of the sphenoid was left to interfere with drainage, have required attention, and the results have been unsatisfactory. The elaborate and trying technic comes at the end of a long, difficult operation. The tendency to hurry is only human. After an operation I have often had a feeling of incompetency that had at least the beneficial result of sending me back to the dissecting room for more practice.

The ligation of the sphenopalatine artery is not so satisfactory as the other ligations that I have developed. The vessel is tied in mucous membrane. It is not isolated. It is sometimes difficult to place the ligature as far laterally as one would wish. Our anesthesia often is not so satisfactory when we begin to work on the nasopharyngeal wall. The solution of the problem may be greater celerity in the earlier and simpler parts of the operation. This should be advanced by the use of clips instead of by ligatures. Nothing makes for smooth, expeditious work, however, as does constant practice. This can be satisfactorily obtained on the cadaver.

TECHNIC FOR LIGATING TERMINAL VESSELS

I have recently developed a transantral technic for ligating the terminal branches of the internal maxillary artery at their entrance to the nose. The sphenopalatine artery is tied here much more satisfactorily. I hope to work out some combination of the two operations that will utilize this ligation for the sphenopalatine and the posterior lateral nasal arteries. Most patients requiring the frontal-ethmosphenoidectomy have infection in the antrums. It is my present plan to attack a one-sided pansinusitis by operating first on the antrum, according to a modified Caldwell-Luc method. In removing the naso-antral wall in the middle meatus, I make an important variation from the classic technic.

This allows me to reach and tie the terminal branches of the internal maxillary artery submucously just at their entrance to the nose. The ethmoid cells in the posterior part of the lateral mass can be opened, and the infra-orbital cells, particularly, can be drained also. The technic described for the sphenoid can be carried out in some cases at that time, if the conformation of the bones gives working space. As soon as the patient is fit again, frontal-ethmosphenoidectomy may be performed. The sphenopalatine artery has been tied and should not give any trouble if the sphenoid technic is carried out, when that has not already been done or has not been properly done at the antral operation.

Patients with pansinusitis have infection requiring the surgeon's attention which is located in many different areas—all difficult of approach. It is not to be wondered at that many operations have been required when an attempt has been made to drain one or a few cells at a time. Often before the succeeding operation has been performed, the good results from the first work have been stultified by accumulation in the cavity in which operation has been performed of pus from sources not yet attacked. Recently a patient consulted me who referred to thirty different operations performed on her sinus. This leads to the disheartening statement not uncommonly heard from the laity that "an operation on a sinus brings endless trouble and repeated operations." Some rhinologists of excellent repute concur in advising their patients against operations on the ethmoid sinus, saying that they are not helpful.

Nevertheless, the medical profession recognizes the significance of sinus infection in the causation of disease. They refer their patients to otolaryngologists to obtain relief from that infection. When other forms of treatment are so frequently unavailing, surgical intervention seems to present some hope. If surgical procedures are to be used, the work must be thorough and fulfil the requirements of surgical work. Dependent drainage is the most important surgical detail to be attained. We have operated on seventy patients, and can say that the results are better than by any methods we have formerly used. An encouraging number of our patients are well or relieved from their symptoms to a great extent. We have had to reoperate on three patients, and some patients have given us much trouble; practically all are better than before the operation.

There is no desire on my part to minimize in this report the difficulties and discouragements attendant on the treatment of patients with sinus disease. All otolaryngologists know the status of work done on the sinus by successful surgeons, and derive but little encouragement from the recital of operative miracles. I believe that we have barely scratched the soil in preparation for the harvest that the future will enjoy, but we are happy if our endeavors cause a seed or two to take root.

MARKED DEAFENED AREAS IN NORMAL EARS*

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As one notes the various forms of normal and abnormal ears as graphically depicted by audiometer readings, many peculiarities recur so often that it would seem that they have some definite meaning. One striking phenomenon seen in a large percentage of hearing graphs is a marked dip in the curve between 1,000 and 5,000 cycles (most often about 3,000 to 4,000). This occurs frequently in both normal and abnormal ears, and its significance is unknown.

It was thought at first that some peculiarity of the telephone receiver in contact with certain ears might cause the losses in hearing near these frequencies, but hundreds of cases have been checked with calibrated tuning forks, and little, if any, discrepancies have been discovered. Moreover, the bone conduction, measured at the same time as the air conduction, often coincides with the latter, thus eliminating, according to present conceptions, any errors due to the presence of unseen obstructive lesions. Obstructive lesions, if present, would moreover show losses at other frequencies. So one may assert that in many otherwise normal ears marked deficiencies of hearing limited to one, two or two and a half octaves of the scale may and do occur irrespective of any detectable defects in the conduction apparatus. The apparently normal ear will be discussed in this paper, and to limit the discussion, a striking example will be taken as the text.

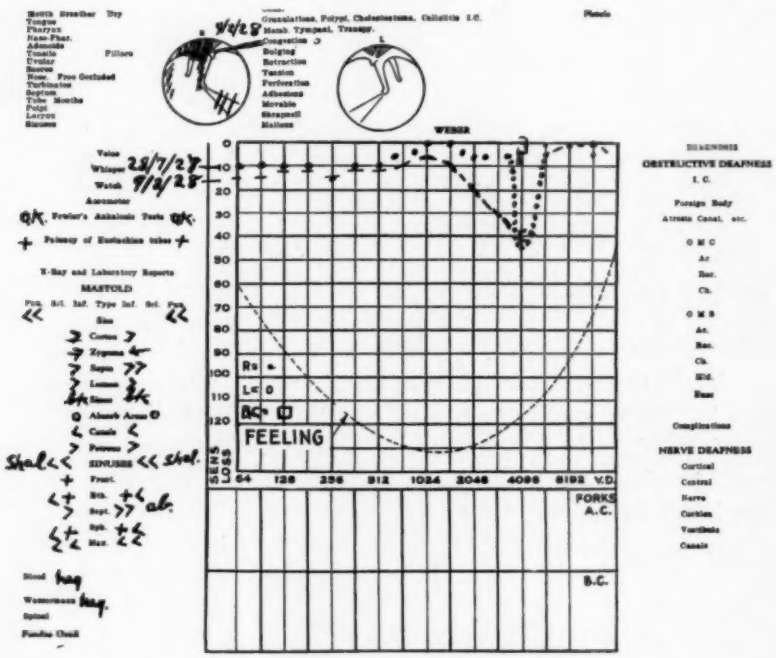
It has been stated that in measuring losses of hearing at intervals of an octave, one might easily fail to note the presence of even marked losses that might occur between the points plotted. This does not often prove to be the case, because the losses almost always extend over more than an octave, and usually from over an octave and a half to two octaves. However, it is certain that with half and third octave intervals as in the IA audiometer I have used for the past six years, none of the variations from normal would have been missed. Over a thousand graphs have been studied with this idea in mind. The markedly deficient tone area in the case considered is the smallest thus far seen. As shown in the graph, it extends from 3,251 to 5,793 cycles.

No defect in hearing was complained of, and the hearing was charted merely as a routine procedure to determine its normalcy on the date of the test. Unless one does this in many cases, it will always be uncertain how and why certain troubles in hearing begin. The observa-

* Read at the Meeting of the American Otological Society, Washington, D. C., May, 1928.

tions were repeatedly checked, and the subject was an accurate observer. There is no doubt of the accuracy of the data.

Each dot on the chart represents a charted observation, the great number being made possible by an attachment to the audiometer whereby the frequency intervals may be tested at intervals of 100 cycles (double vibrations). This search demonstrated little deviation from the curve drawn between the three points originally plotted at 3,254, 4,096 and 5,793 cycles. The hearing of the left ear was high normal, and that of the right ear was also average normal except between the critical fre-



In the chart, the 1927 test shows the marked deafened area in the right ear; 1928 test shows the slight congestion in the attic and along the malleus and its effect on the canal of hearing in the right ear.

quencies. Bone conduction was almost identical from both mastoids, except when a masking noise was applied to the left ear, when it dropped exactly 30 sensation units to a point only 5 sensation units less than the air conduction curve. This illustrates the necessity of using a masking noise apparatus when testing bone conduction. As I have repeatedly insisted, the ear not being tested must be made deafened to the testing intensity, in order to assure a test limited to the opposite ear; otherwise the ear opposite to that apparently under test will be the one that partly or wholly hears the sound.

There are four explanations of these limited deafened areas which theoretically seem logical, and they are as follows: The curve dip is due to (1) some limited central or cortical lesion in an area presiding over these frequencies, (2) a defect in the nerve fibers to the tiny section of the basilar membrane which normally detects these frequencies, (3) a defect in the terminal nerve apparatus (Corti's organ) or (4) an anti-resonance somewhere in the conducting mechanism. In other words, the frequencies are filtered out before they reach the end-organ in the cochlea.

If the variation in hearing occurred in both ears and in similar areas, it would seem that some equal bilateral nerve or unilateral central or cortical cause must govern, but although equal bilateral marked depressions often occur, they are seldom seen in normal ears. The hearing in a normal ear usually varies somewhat from that of its fellow at these frequencies, but it is difficult to understand how the central or cortical influence can be proved to be the cause of the phenomena.

The area of quiet is so clearcut and limited that at first sight it is suggestive of a defect in one or two small terminal nerves, but the plotted points show that the trough is very sharp, and, although precipitous on both sides, is not squared, but rises almost perpendicularly to the upper and lower frequency rims. How could a block or bundle of defective nerve fibers operate thus? They could not, because the whole bundle would probably be deficient to the same extent, and the fibers leading to the areas of the frequencies immediately adjacent to the points of greatest loss would not on both sides be so regularly and progressively affected—indirectly as the distance from the bottom point of the depression. If the defect were wider, as is usually the case when it occurs in both normal and abnormal ears, and if the trough of the wave were level and its sides equally precipitous, I should favor the theory of a defective nerve or nerve group, occasioned possibly by some swelling or pressure, self-contained.

A defect in Corti's organ is strongly suggested. Imagine the membrana tectoria deficient or thinned in a tiny section or burdened with a large vessel so that imperfect or little contact obtained between it and the hairs. Imagine a deficient arterial blood supply to the area or an embolus in the terminal vessel to the part or a blocking of the return flow therefrom. The branching point of the distal and proximal branches of the cochlear artery occurs approximately at the level of the basilar membrane, which corresponds to the area of the frequencies affected in these cases. It may be that the point of branching influences the blood supply adversely in some ears; that is, causes, as it were, a deaf spot on Corti's membrane by interfering with the perfect nutrition of, or by crowding out, a few of the nerve fibers in that region. It may be that these defects vary as to frequency even in normal ears, because

of the different places and manner of branching of the cochlear artery. If disease subsequently occurs near this spot in such an ear, the deafness is likely to spread from the greatest trough depression to one or both sides. It is possible to imagine many causes, but at present, it is impossible to prove their correctness. Only sectioning the ear, after examining it carefully during the life of the patient, will clear up the mystery if the cause of the deafness lies in the cochlea.

Antiresonance is some resonator or filter action which eliminates its own attuned vibration periods from any sounds passing. If a complex sound is passing along a solid or hollow system of conductors (i.e., a rod, a tube or the external meatus and middle ear conducting chain) and a resonator (any acutely tuned vibrating medium such as a Helmholtz resonator, or a free reed mechanical resonator) is placed in contact with the vibration path, the sound will travel to its destination unaltered, except that the frequency coinciding with the resonator frequency will be absorbed by the resonator, and thus taken out of the original sound, and fail to reach the terminal element of the system. Any number of resonators may be arranged along a tube, or a rod or system of rods or levers, and each resonator will filter out its own frequency. In connection with the middle ear mechanism there are several possible resonators, such as air spaces of differing sizes and free or thickened tissues.

It is noteworthy that the tone deficiencies under discussion always occur within the higher frequencies (about 4,000 double vibrations), and it is possible that they could easily be caused by resonance absorption as suggested. Some reasons for this belief are: (1) the clearcut limitations of the deafening, (2) the sharp cut off from the normally heard frequencies to either side, (3) no impedence lesions visible to the eye or detectable by the usual functional tests and (4) the changes observable when an ear with such deafened areas undergoes even a slight congestion.

In the case under discussion, one day a slight feeling of blockage and tension occurred in the ear. Otoscopy showed congestion in the attic and along the malleus and slightly behind it on the drum membrane. The audiogram showed that the dip (by air and bone) was the same as before, and that the lower frequency side, instead of being precipitous, sloped away at an angle of approximately 45 degrees to 2,048, where it was normal. This slope was plotted at intervals of 100 cycles and found to conform with the 45 degree curve, without any variation of more than 5 sensation units. It appears that such a change in curve could be brought about by mechanical means, by altering the vibrating characteristics of the conducting chain and especially by lessening the effect of the resonator filtering at the frequencies near 4,096 by spreading this to include also, but in diminishing effect, the frequencies between 4,096 and 2,048 as shown in the graph.

The effect on the frequencies outside of the dip was practically nil; hence there was little interference with the mechanism of the middle ear, except that the slope and width of the dip was changed on its lower side. Was this due to the introduction or withdrawal of other filtering effects? If not, what was the cause?

Within a few days, the congestion in the attic disappeared, and the graph resumed its original slope. The proportionate loss by bone conduction was not altered. This indicates that sound transmitted by way of the cranial bones was not perceptibly influenced by the changed condition of the middle ear, and that bone conduction transmission was shunted around the interference, but was still affected by the filtering agent to the same extent as the air-conducted sounds.

COMMENT

The threshold of hearing in the left ear was the same as that in all normal ears—a little indefinite without repeated search—whereas in the right ear (near 4000) there was no hesitancy in the sensing of the exact point every time it was reached; in other words, the sound was definitely absent or very apparent to the observer. The threshold appeared suddenly.

Comparative loudness showed that the loudness of tones (near 4,096) of from 10 to 25 sensation units above the threshold in the left (non-affected) ear appeared the same loudness as 5 sensation units above the threshold in the right (affected) ear.

At frequencies just below the brims of the dip, the ear heard all the frequencies double. The change to a clear and single tone took place at 3,400 and 4,700 cycles.

These observations suggest static friction somewhere in the conduction path; probably not in the middle ear, because it would affect several other frequencies, and these would then also be lowered, which was not the case. If the friction occurred in the cochlea, it would seem that it must be in Corti's organ, because otherwise it would lower all the lower frequencies; if in Corti's organ, it would possibly be looseness or altered coaptation in the rods of Corti or the hair cells and tectorial membrane.

Is it possible that there are many normal ears like this? Is this a normal ear? Is there such a thing as a normal ear? Some day we shall find out!

114 East 54th Street.

PHRENOSPASM AND CARDIOSPASM IN MEGA-ESOPHAGUS

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There is still much discussion as to whether the spasm in cases of so-called "cardiospasm" with large dilatation of the esophagus is located at the diaphragmatic region or at the cardiac region. My personal researches on the subject allow me to draw certain conclusions, which seem to throw a new light on the mechanism of the spasm.

Jackson [1] was the first to suggest that a "phrenospasm" occurred in these cases; his studies were made with the esophagoscope.

Guisez,[2] in his book on the subject, spoke at one time of "phrenospasm" and at another time of "cardiospasm," but he does not seem to be any more in favor of one theory than of the other. Sargnon,[3] although he is a partisan of the congenital theory of Bard, does not even discuss the seat of the spasm.

The question has, however, been thoroughly discussed by English specialists.

William Hill [4] said: "Many doctors, surgeons and radiologists say now that the stenosis begins at the cardia. We know through experiments with the roentgen ray that it is caused by the contraction of the diaphragm."

The question of pathogenesis is far more interesting. Almost all the authors admit that at the beginning some nervous element is a factor; this element is supposed to be a stimulation of the vagus, resulting in hypertonicity.

Shattock [5] said that the esophagectasia is due to the incoordination of the nervous influx transmitted to the vagus in swallowing; this influx normally brings about the contraction of the tube and an active dilatation of the cardia. This is also the opinion of Rolleston and Hurst.

Other authors (Henke and Morell Mackenzie) have suggested a primary idiopathic condition. But this explanation, though plausible and defensible for congenital dilatation of the esophagus, especially after

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1. Chevalier Jackson: Endoscopie, Paris, Gaston Doin, 1914.

2. Guisez: Diagnostic et traitement des retrecissements de l'oesophage, Paris, Masson et Cie, 1923.

3. Sargnon: Cancer de l'oesophage, Paris, Gaston Doin, 1927.

4. Hill, William: Meeting of the Section of the Laryngology, December, 1918, London.

5. Shattock: Proc. Roy. Soc. Med., 1916-1917.

the case published by Van den Wildenberg,[6] cannot be applied to acquired dilatations.

Having collected the opinions of various authors, I made some researches to clarify the matter. I began with the radioscopic study of the normal esophagus. The subject of the experiment was placed before a fluoroscopic screen and asked to swallow an opaque substance. This would have demonstrated nothing, had not a small dose of atropine, which I thought would paralyze the esophagus and the cardia, first been administered hypodermically. One milligram of sulphate of atropine was injected under the skin, and after about thirty minutes the examination was made. I was greatly surprised when I found that, instead of patency of the cardia, a spasm was produced.

Repetitions of this experiment always gave the same result. At times a threadlike opening was found sufficient to allow a part of the esophageal contents which had accumulated above the diaphragm to pass into the stomach. I could therefore draw the conclusion that the atropine had had rather a spasmodic, instead of a paralyzing, effect on the phrenocardiac musculature, for there was a portion about 1½ or 2 inches (3.77 to 5 cm.) long that was constricted and drawn down toward the left.

These experiments, which will be published in detail elsewhere, pointed out the mistake that has been made until now, namely, that whereas the cardia has been considered to be innervated exclusively by the vagus, the sympathetic innervation must now be taken into consideration. Having arrived thus far, it was easy to conclude that at the beginning of a dilatation of the esophagus, the spasm was not caused by excitement of the vagus, but rather by a sympathetic excitement or a vagal hypotonicity.

Later, I was fortunate enough to be able to make an examination also in several cases of esophageal dilatation, and in these cases nothing was found but a spasm at the cardia. The opaque mixture accumulated at the cardia, and when the subject of the experiment swallowed some liquid, the accumulated opaque mixture fell in a lump into the stomach. This time the threadlike constriction was no longer perceived, but only a spot definitely located just below the dome of the diaphragm.

It was necessary to find an explanation of this phenomenon, for whereas a phrenospasm was found in normal subjects, only a cardiospasm and considerable dilatation was found in the patients with esophagectesia.

In the initial stage, that is, at the beginning of the dysphagia, when it is still intermittent, there is a phrenocardiospasm. Thus, in the work of Hill [4] the illustration, figure A, is altogether correct, for a slight dilata-

6. Van Den Wildenberg: Pan Americain Congres of Otolaryngologists, May 1927.

tion of the esophagus with a phrenocardiac spasm can be perceived. This spasm lasts as long as its cause, the dysphagia progresses, and the residue of food in the esophagus increases. It is then that the mechanical influence begins to act. The material lying just above the spasm becomes more abundant, and its weight presses down more and more, so that the phrenic muscles become tired and a relaxation of the phrenic spasm is thus produced, leaving only a cardiac spasm. This is the reason why in the roentgenograms an opaque mass in the form of an inverted cone is perceived above the cardia. This mechanical factor is augmented by the repeated peristaltic waves which are broken against the spasm and help thus little by little to tire it out. The roentgenograms in the book by Guisez [2] are an evident proof of the theory I have advanced.

One might think, like the esophagoscopists, that perhaps the spasm is caused by an inflammatory influence on the mucosa of the esophagus, and that the inflammation is increased by the stagnation of food. But if this inflammation were the predominant factor, the phrenospasm would tend to increase rather than to reflex. Besides, I have made an examination in several cases of dilated esophagus, with the help of esophagoscopy, and have not always found inflammatory lesions; in fact, the number of cases without inflammation was larger than that with inflammation. This seems to prove the predominance of a nervous element in the causation of spasm.

CONCLUSION

This study shows that one must renounce the idea of the predominance of the vagus in the pathogenesis of this condition, and admit rather a sympathetic predominance, or possibly a hypotonicity of the vagus; and secondly, that at the beginning of an acquired dilatation of the esophagus, a phrenocardiospasm exists, which is later changed into a cardiospasm through a mechanical influence amplified by peristaltic action.

ANGINA LUDOVICI

AN ANATOMIC AND CLINICAL STUDY *

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Ludwig's angina is a phlegmonous process arising from infections within the floor of the mouth and localized in a definite anatomic space.

This space is secured by boundaries, having as its floor, the mylohyoid muscle; as its lateral walls, the bodies of the mandible; as its posterior wall, the muscles which unite to form the base of the tongue and the deep part of the submaxillary gland, and, as its roof, the tongue and the mucosa covering the floor of the mouth.

The condition, being thus limited, quickly induces alarming local symptoms, which make it one of the most dreaded diseases. If treatment is not prompt, in the early stage, the mortality is high; it is given by various authors as approximately 50 per cent.

Von Ludwig,[1] after whom the disease is called, described a case, characteristic in every detail, of a rapidly spreading infectious process confined in this anatomic space. His was so typical of a certain group of cases reported by other writers, and, at the same time so different, from the standpoint of treatment and prognosis, from some other disease conditions about the mouth and throat so often confused with it, that it is desirable to retain the name and consider it a distinct clinical entity.

The origin of the infections which develop into Ludwig's angina is within the lower gingival borders, usually around the teeth and within the floor of the mouth. This was determined by a study of the lymphatic drainage of the regions about the mouth in a series of animal experimentations made by me.

The infectious process in Ludwig's angina may be confined to either or both sides of the sublingual space, or it may appear about the midline. When it is confined to either side, the swelling forces the tongue upward and toward the opposite side. When it is on both sides simultaneously, or about the midline, the tongue is forced upward toward the roof of the mouth. Coincidentally there is swelling and edema of the mucosa of

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* Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society.

1. Von Ludwig, W. F.: Ueber eine in neuerer Zeit; Wiederholt hier vorgekommene Form; von Hansenlzundung, Med. Cor.-Bl. d. württemb. ärztl. Ver. Stuttg. **6**:21, 1836.

the floor of the mouth over the area corresponding to the infectious process. This edema, as in one of my cases, is often so marked as to resemble a secondary tongue.

Another prominent symptom of the disease is a boardlike swelling, externally, over the region involved. This swelling does not have any points of fluctuation, a fact easily explained by the absence of pus formation outside the muscle floor of the mouth which is under great stress from within. Some authors, notably Thomas [2] who has made an extensive study of this subject, believe that the primary infection in Ludwig's angina is in the submaxillary region outside the floor of the mouth and subsequently reaches the sublingual space by a process of retrogression. Their contention in this respect will not harmonize with the absence of fluctuation, which they concede, since pus formation in this region would easily show fluctuation, it being superficial. In addition, Thomas believes that a case should not be diagnosed Ludwig's angina until the process has invaded the sublingual space; however, at operation, he found pus in the sublingual space after piercing the mylohyoid muscle, and did not find pus superficially—another fact which would disprove the theory that the primary infection is in the submaxillary space.

In order to give a clearer understanding of the relationship between the symptoms of the disease and the anatomy of the involved parts, the following anatomic descriptions are submitted:

If the tongue, mucous membrane, loose tissue and geniohyoid muscles are removed from the floor of the mouth of the cadaver, a complete muscular diaphragm formed by the mylohyoid muscle will be found. This is a fanshaped muscle attached at the handle end to the hyoid bone and at the fan end to the mylohyoid ridge on the inner surface of the mandible. The mylohyoid ridge extends from a point 1¼ inches (0.6 cm.) below the gingival margin in front to a point on a level with the gingival margin at its posterior extremity, ½ inch (0.2 cm.) behind the last molar tooth. The position of this ridge is important in that the mylohyoid muscle elevated posteriorly and dipping down to the point of the chin anteriorly forms a more complete pouch toward the front of the sublingual space, and at the same time this space is more completely separated from infections more posteriorly in the neck (fig. 1).

The anterior fibers of the muscle converge from their mandibular origin and are inserted in a median raphe. The posterior fibers are attached to a strong fibrous band which reaches from the central portion of the body of the hyoid to the great cornu of the same. In doing so, the fibrous band passes over the insertion of the subhyoid muscles with the sheaths of which it is intimately connected (fig. 2).

Posteriorly the floor of the mouth is limited by the hyoglossus and styloglossus muscles and the deep part of the submaxillary gland, as its body bends around the posterior border of the mylohyoid muscle (fig. 3). There is also a strong fascia sheath extending from the inner surface of this gland to the side of the tongue (fig. 2).

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2. Thomas, J. J.: Ludwig's Angina: An Anatomical Clinical and Statistical Study, Univ. Penn. M. Bull. **20**:27, 1908.

The hyoglossus muscle, according to Cunningham, arises from the body and great cornu of the hyoid bone and is directed upward and forward to be inserted into the side of the tongue, its fibers interlacing with the fibers of the styloglossus muscle (figs. 2 and 3). This muscle advances forward and medially to be inserted into the side and the inferior surface of the tongue, its fibers spreading out to

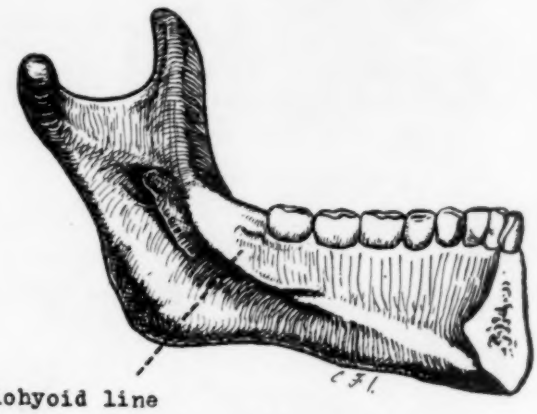

Fig. 1.—Mylohyoid ridge elevated posteriorly and dipping down to the symphisis menti anteriorly.

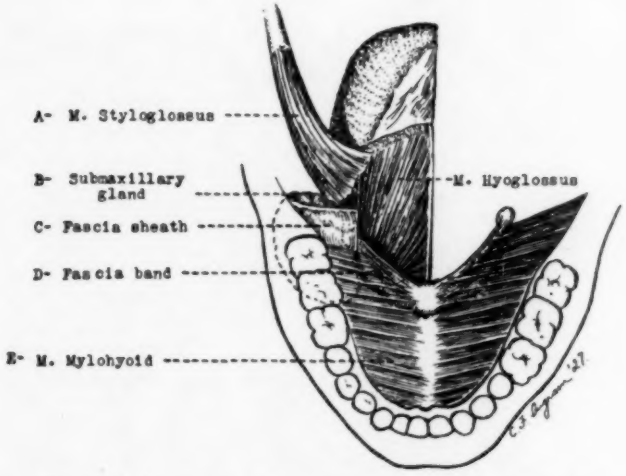

Fig. 2.—Diagram of area formed by the mylohyoid muscle.

decussate with those of the glossopalatinus and hyoglossus muscles beneath the submaxillary gland and mucous membrane of the tongue.

The submaxillary gland plays a prominent part in the posterior limitation of the sublingual space. This gland is divided into two parts; a superficial part lies outside the mylohyoid muscle (fig. 4), and bends around the posterior border of that muscle, where it becomes the deep part of the gland. This part is wedged between the mylohyoid and hyoglossus muscles (fig. 5).

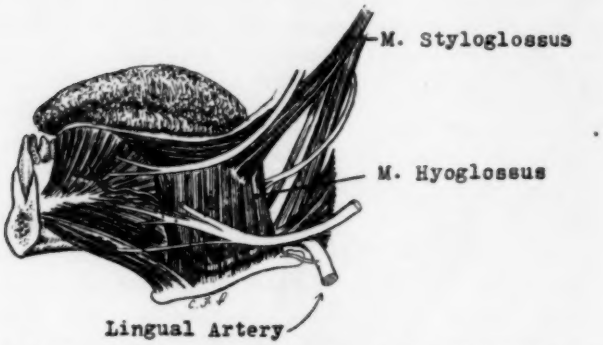

Fig. 3.—Muscles which form the posterior boundary of the sublingual space.

Fig. 4.—The external part of the submaxillary gland.

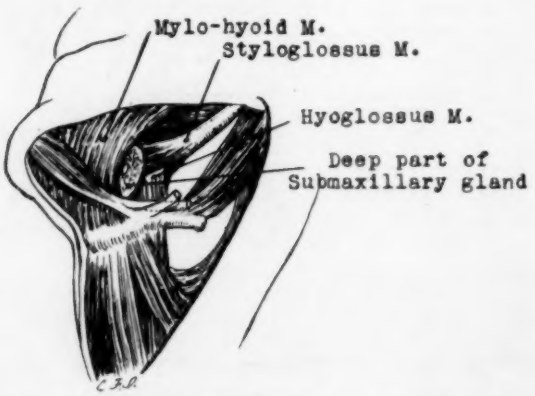

Fig. 5.—The internal part of the external gland after it has been around the posterior border of the mylohyoid muscle.

Thus, there is an anatomic pouch which is capable of greatly influencing the course of an infection originating in any of its contained structures. The weakest point in the walls of this pouch is to the inner side and below the deep part of the submaxillary gland. It is through this space that the lymphatic glands of the tongue, except of its tip, and the other structures in the floor of the mouth, find their exit to the deeper lymphatic glands of the neck.

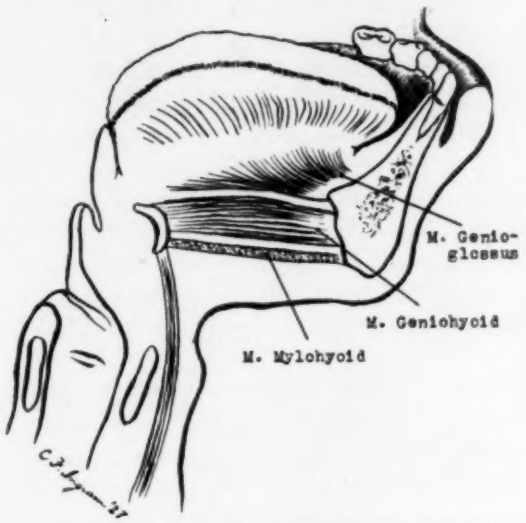

Fig. 6.—The relationship of the mylohyoid muscle to the other muscles inside the mouth.

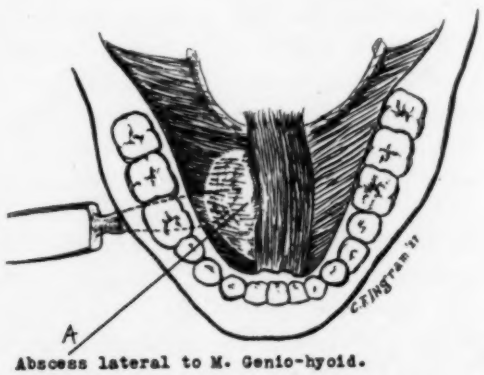

Fig. 7.—The relationship of the geniohyoid muscle to the floor of the mouth; also the influence this muscle has on the position at which an abscess may form.

The geniohyoid muscles cross the floor of this space postero-anteriorly near the midline (fig. 6). These muscles are loosely attached to the mylohyoid muscle on the bottom and the geniohyoglossus muscle on the top, and have some influence as to the position at which an abscess forms. The abscess may form to the outside of the muscle as seen in figure 7, and, according to available reports of cases, there is where it does form in a majority of instances. On the other hand,

the abscess may form between the geniohyoid and the mylohyoid muscles in the midline (fig. 8), or it may form between the geniohyoid and the geniohyoglossus muscles, in the midline (fig. 9).

A series of experiments, in which India ink was injected under the mucosa in various parts of the mouth and fauces of dogs, demonstrates the course of the lymphatic drainage of those parts.

It was found that ink injected under the mucosa within the lower gingival borders, but not penetrating the mylohyoid muscle, and also that injected in the peritonsillar space, coursed backward and infiltrated the deep cervical gland, internal and slightly posterior to the sublingual gland and lying alongside the carotid sheath—the deep cervical gland. Ink injected under the mucosa inside the lower gingival border near the front teeth, and also the ink which penetrated the mylohyoid muscle at the time of injection, coursed backward outside the floor of the mouth to the small lymph glands situated on the superficial surface of the submaxillary gland. Not in any case was the submaxillary gland infiltrated.

These experiments indicate the courses which infections about the mouth might be expected to pursue. Infections arising around the inner side of the lower teeth and gums, except at the front, course down into the tissues of the sublingual pouch where an inflammation begins and finally develops into a sublingual phlegmon, or Ludwig's angina. Not all infections thus arising, however, pursue this simple course. Some of them pass on posteriorly to the deep cervical glands without producing any sublingual inflammation and subsequently enter the general circulation. This was well illustrated in one of my experiments in which a virulent strain of streptococci was injected under the tongue of a dog. Sublingual evidence of the disease was not observed, but, after one week, a general septicemia developed, which was manifested by numerous small abscesses covering the entire body. Other infections produce Ludwig's angina, and then pass on posteriorly to the deep cervical lymph nodes, giving rise to far-reaching complications. Likewise, the experiments indicate that infections in the superficial structures outside the floor of the mouth, that is, under the chin and in the superficial submaxillary region, may have their source inside the mouth near the lower front teeth, in the gums and around the teeth on the external side or outside the midgingival border. In addition, they indicate that infections around the tonsils do not cause Ludwig's angina. It is doubtful if the tonsil, or any structure behind the styloglossus ligament, is ever the primary source of Ludwig's angina. Nothing in these experiments sustains the theory that the primary infection in this disease is in the submaxillary region. Such a belief could not be justified on the grounds of the lymphatic drainage. The assumption that it travels by a retrograde

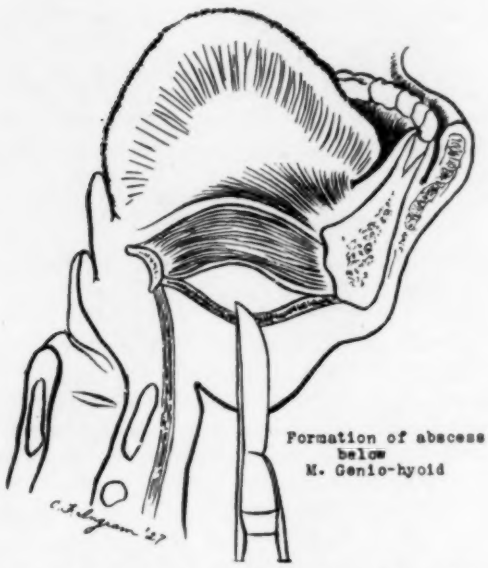

Fig. 8.—The position of an abscess deep above the geniohyoid muscle.

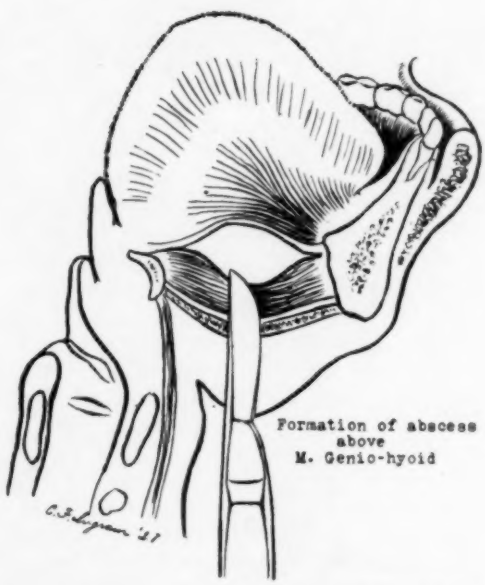

Fig. 9.—An abscess between the mylohyoid and geniohyoid muscles in the midline.

course by proceeding upward into the floor of the mouth against the force of gravity and without the aid of muscle and fascia planes is without parallel in any known pathologic process. The arterial flow is the only agent offering assistance to such a course. Furthermore, the idea of a primary infection in the submaxillary region predicates an idiopathic origin—an idea that is more and more growing obsolete in medical parlance.

REPORT OF CASES

I have had four cases of Ludwig's angina under my care within the last two years. Histories of these cases are as follows:

CASE 1.—S. J., a man, aged 35, in good physical condition, developed a swelling under the middle of the right part of the lower jaw. A pyorrhea pocket around the first right lower molar was discovered by his dentist who advised that it should be opened. He did not seek further medical aid until the sixth day, although the condition had continued to grow worse. I saw him at that time, when there was a great swelling over the right side of the face and neck, forcing the head toward the left side. It extended from the ear to the tip of the chin and from the hairline in front of the ear to below the clavicle. The swelling was hard and boardlike without any point of fluctuation. The point of greatest swelling and tenderness was under the right side of the lower jaw, midway between the angle of the jaw and the point of the chin.

The tongue was raised toward the roof of the mouth, making examination of the fauces difficult; this could be performed only by pushing the tongue to one side, and not by pressing it down in the middle. There was considerable edema of the uvula, the right side of the palate and the anterior pillar of the corresponding tonsil. The mucosa under the right side of the tongue was so edematous and red as to simulate a secondary tongue.

The patient was having difficulty in breathing, whether this was caused by the saliva, which he was unable to swallow, or by the edema encroaching on the larynx could not be determined. When he attempted to swallow water, it would regurgitate through the nose. The temperature was 103 F.; pulse rate, 120, and respirations, 20. The leukocyte count was 12,450; polymorphonuclears, 88; large mononuclears, 5; small mononuclears, 7. An incision 2 inches (5 cm.) long was made at the level of the lower border of the jaw. The tissues showed great resistance on being cut. The incision was then carried beneath the jaw and extended through the muscle forming the floor of the mouth, where an abscess was entered at the middle of the jaw, and a drachm of thin flocculent pus was evacuated. Cultures of this pus showed streptococci. A small incision was made below which converted the wound into the *T* type. The wound was swabbed with mercurochrome. Two rubber tissue tubes were inserted. Blood cultures were made on three successive days without results. A tracheotomy set was provided. Antistreptococcic serum was administered. Some improvement was noted on the second day, and the temperature became normal on the third day after the operation. Recovery was complete in two weeks.

CASE 2.—Mrs. K., aged 35, who was in rather poor health, had a large swelling beneath the chin between that point and the hyoid bone. The swelling was hard without any point of fluctuation. No area of infection was located. The mucosa

under the tongue was edematous, and pushed the tongue up toward the roof of the mouth. She complained of severe pain, and swallowed with great difficulty. The temperature was 102 F.; the pulse rate, 100, and respirations, 20.

An incision was made in the midline between the point of the chin and the hyoid bone through the mylohyoid muscle. Forceps were inserted into the sublingual region. About 2 cc. of thick, creamy pus was discharged. Streptococci were found in the culture of the pus. The wound was irrigated with mercurochrome. One small rubber tube was left in the wound and stitched to the skin. The temperature subsided to 99 F. after evacuation of the pus. The next day the temperature rose to 101 F. The drainage was slight. The edema in the mouth increased, and the abscess was beginning to point under the left side of the tongue. The abscess was incised inside the mouth and thick yellow pus was freely drained from it. The temperature returned to normal on the third day, and the patient was discharged two days afterward in an improved condition. Subsequently she went on to an uneventful recovery.

CASE 3.—J. C., a man, aged 34, in excellent physical condition, developed pain and swelling under the tongue six days prior to his entrance to the hospital. The symptoms increased, and at the end of three days he was unable to eat and could swallow water only with the greatest difficulty. A source of infection could not be located.

Examination on his entrance into the hospital revealed the following:

There was a marked swelling under his tongue, pushing it up against the roof of his mouth; it could not be pressed down in the middle. When the tongue was pressed to one side, the pillars of the fauces were found to be edematous. The voice was gutteral. There was a marked swelling under the chin in the midline between the chin and the hyoid bone. The swelling was hard and board-like without any point of fluctuation. His temperature was 100.4 F. and his pulse rate was 90. The leukocyte count was 10,800. The general examination otherwise revealed negative observations.

The operative technic was as follows: After the surface of the neck was sterilized, 1 per cent procaine hydrochloride was injected in the skin between the chin and the hyoid bone. A large hypodermic needle was inserted straight into the sublingual space. This pierced a tough membrane at a depth of 2 inches (5 cm.), where a cavity was entered. Withdrawal of the plunger of the syringe revealed about 1 drachm of thick flocculent pus. An incision 2½ inches (6.3 cm.) deep was made in the same direction, entering a purulent cavity inside the hyoid muscle. The cavity was flushed with 5 per cent mercurochrome and packed loosely with iodoform gauze.

The culture of the pus showed: (1) nonhemolytic streptococcus; (2) *Staphylococcus aureus,* and (3) diphtheroids.

The patient's condition improved steadily for ten hours, at which time the drainage stopped; the temperature again rose in twenty-four hours to 101 F. The packing was removed, the cavity was flushed with mercurochrome, and two small rubber catheter tubes were inserted for drainage. Following this, the drainage was free and his condition steadily improved. On the seventh day, he was discharged as cured.

CASE 4.—S., a man, aged 35, was seen by me on the fifth day after a dental scraping of an old tooth socket of a second right lower molar. Two days later, a tender swelling appeared under the tongue opposite the scraped tooth socket and continued to grow worse.

Examination revealed a swelling under the right side of the tongue, half the size of an olive, where the mucosa folded over from the tongue to the mandible. Redness and swelling of the mucosa extended on that side to the frenum. The tongue was displaced upward and pressure on it caused pain. It was evident that there was an abscess inside the floor of the mouth, infection having started from the tooth socket. The patient was sent to a dentist to have the abscess drained. This was accomplished by probing down from the field of the operation and enlarging the opening. One drachm of thick pus was evacuated, and the patient made a prompt recovery.

LUDWIG'S CASE

In 1835, von Ludwig [1] reported a case of gangrenous induration of the neck and gave a description of a certain type of inflammation of the mouth and neck that has become classic. His case on the sixth day of illness was described in part as follows:

The swelling had progressed with clearly defined stony hard margins as far as the point of the chin, while erysipelatous redness of the pharynx with difficulty in swallowing, and swelling and redness of the floor of the mouth under the tongue, were noted on examination.

On the seventh day:

The floor under the tongue had become a deep dark red in color, and was extremely hard, while a swelling provided with a calloused margin of a degree of hardness similar to that encountered in scirrhous growths extended around the internal border of the inferior maxilla. There was added difficulty in swallowing. Movements of the tongue and opening of the mouth were still further restricted.

From the seventh day, the local symptoms increased in severity.

The tongue was pressed backward and upward by the calloused ring and the hard swelling beneath that organ; speech was indistinct, with a croupous sound, and the patient was hardly able to open her mouth sufficiently wide to admit the little finger. From the tenth day onward the swelling began to subside and fever gradually, but slowly, diminished. First, the sterno-decido-mastoid could again be freely palpated; then, the larynx became free, the mouth opened more easily, the calloused swelling in the mouth became softer, the base of the tongue less red, and the tongue freer in movement, with consequent improvement in speech. Later on the swelling receded from the right side of the neck in the direction of the chin. As previously remarked, softening did not occur simultaneously, but completely soft parts were found in contact with still extremely hard margins of swollen areas. Even in presence of this phenomena of retrogression the cellular tissue of submaxillary and sublingual glands (but not the glands themselves), remained hard. At the same time and within the same period, hardness in the mouth disappeared entirely and the cutaneous eruption dried up. Only the tissue already established was maintained in a state of suppuration by means of escharotics, etc. Hardened areas, which remained, now became gradually softer at their points of origin and the patient again felt well and strong. Final traces of hardness were still palpable at the end of the third week of illness.

CLASSIFICATION

A few months ago, in a questionnaire sent to the members of the American Otological, Rhinological and Laryngological Society, I noted that of the 104 who answered, forty-five (or 43 per cent) were of the opinion that Ludwig's angina should be considered a clinical entity; twenty-nine were of the contrary opinion, and thirty-eight had not had any experience on which to base an opinion. A history of the earlier writers on the subject shows a tendency toward classification.

There are a number of writers on this subject, notably, Sir Felix Semon, who think that there should not be any classification of the various infections around the throat and mouth, since it cannot be shown that their bacteriology and pathology are different. It is my opinion that a classification on an anatomic basis is not only desirable, but amply justified from both a historical and a therapeutic standpoint. Diseases that are not accurately described cannot be uniformly treated. If classification of these diseases according to an anatomic basis would lead to a more established form of treatment, this is desirable. The bearing of classification on the treatment of the disease will be commented on later.

HISTORY

Reference to inflammatory conditions about the throat and neck were made as far back as the time of Hippocrates. The conditions were referred to under the term "cynanche," which literally means "dog choking." Under this designation were included the acute conditions which are today classified under the various headings of quinsy, edema of the glottis, retropharyngeal abscess, peritonsillar abscess, sublingual phlegmon and Ludwig's angina.

Hippocrates gave an accurate description of the tongue as it is found in sublingual phlegmon, of which he says: "Changes from broad and becomes round; its natural color turns to livid; from a soft consistence, it becomes hard; instead of being flexible, it becomes inflexible; so that the patient would soon become suffocated, unless speedily relieved."

A further bibliographic study of Ludwig's angina made by me was submitted in my original paper in which a full description was given of the cases that have been identified with this diagnosis. A total of 201 cases were reviewed in the literature. It was seen by the descriptions that ninety-two of this number were not sublingual involvements and should not be classified as cases of Ludwig's angina. The remaining 109 conformed to the description of a typical sublingual infection and should come within the scope of this paper. The ninety-two other cases could be classified into two groups, as follows: (1) those in which the infection was in the pharyngeal structures primarily, and spread to structures lower down in the throat secondarily; (2) those in which the infection was in the superficial strata of the neck.

ETIOLOGY

The bacteriology in all the clinical types of cases reported in the series studied by me is strikingly similar.

The bacteria predominated, according to the number of cases in which they were reported, in the following order: streptococcus, 9; streptococcus and staphylococcus, 7; mixed bacteria, 4; staphylococcus, 3; micrococcus, Salivarius-Biondi, 2; staphylococcus and diplococcus, 1; pneumococcus, 1; *Bacterium ferruginum,* 1; *Bacillus septicus,* 1.

In my series of cases, the bacteria demonstrated by culture from the pus obtained from the abscess were as follows: streptococcus alone in cases 1 and 2, and in case 3 there was a mixed infection of nonhemolytic streptococcus, *Staphylococcus aureus* and diphtheroids.

The same type of infecting organism was found whether the infection was located in the pharynx, sublingual tissues or deep structures of the neck.

Lockwood [3] described a bacillus obtained from the walls of a putrid abscess of the lung which he said was morphologically the same as that found in Ludwig's angina. The bacilli had slightly rounded ends and are about 1 mm. thick and from 3 to 6 mm. long. They did not contain spores, and they multiplied by fission, being often seen in twos or in short chains. The bacilli were numerous where the connective tissue was most abundant as, for instance, in the adventitis of the pulmonary vessels, this fact suggesting that they were anaerobes. They shunned the blood stream and were found distributed along courses parallel with the muscle fibers. He reported a case in which he and Dr. Addison succeeded in demonstrating *Bacillus septicus* by soaking the sections for many days in Czenzynke's solution. They were found in the sections of the deeper edematous muscles and cellular tissues of the floor of the mouth and of the neck.

Some characteristics of *Bacillus septicus* are: rapid growth, shown by the swift spread of the disease; abhorrence of oxygen, shown by its passage along the lymph paths; gas producing power, shown by the emphysematous crackling, and the noxiousness of its ptomaines, shown by the poisonous condition of the patient. Steinberg says the bacillus often causes an inflammatory edema without producing emphysema. Lockwood also describes a case in which the bacillus was demonstrated in the lymph paths and in the inflammatory exudation.

Eugen Frankel described another bacillus—*"Bacillus phlegmonous-emphysematosae"*—which resembles *Bacillus septicus,* except that it is motionless. It was found in four cases of phlegmon.

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3. Lockwood, C. B.: Conveyance of Infection by Contiguity: Septic Pneumonia; Angina Ludovici with Bacillary Septicemia; Local Infection of Angina Ludovici, Lancet **1**:733, 1895.

Burge, in a case of emphysematous cellulitis (gas phlegmon), found *Bacillus coli-communis* and staphylococcus and proteus together.

Rosenbach observed the coincidence of pyogenic cocci and *Bacillus septicus.*

Gartner described a gas producing bacillus which Klein considered identical with *Bacillus coli-communis.*

Lockwood thought that when Ludwig's angina was produced by *Bacillus septicus,* or when this bacillus was a factor, it was associated with other organisms of oxygen consuming propensities.

PATHOLOGY

The tissue reactions to the infecting agent in all types of cases reported as Ludwig's angina show a striking similarity, whether the lesion is in the pharynx, in the region of the larynx, in the floor of the mouth or in the external strata of the neck—namely, a marked edema and rapid spread of the inflammatory process, with an unusual collection of effusion, serous, purulent, fibrinous, hemorrhagic or gangrenous, in the intercellular spaces. If one accepts Lockwood's theory that *Bacillus septicus* may accompany any deep infection about the neck or chest, it is to be expected that emphysema may develop in any of the types of cases reported and that it would not distinguish one type from another.

Sir Felix Semon observed that the micro-organisms causing the various infections about the throat and neck "are interchangeable in the same sense that not one of them produces one form of inflammation and another, another form of inflammation, but that they may all produce different forms of inflammation, according to their quantity, virulence, and to the nature and condition of the tissues of the body they invade. In other words, there is no pathologic organism which always produces a serous exudation and none that always produces a purulent one."

Guerini[4] called attention to the difference between a sublingual phlegmon and a sublingual abscess, the former being a generalized phlegmon, and the latter being a local phlegmon with abscess formation. When it is considered that in the cases reported many of them, with similar clinical manifestations, gave different results when operation was performed—depending, no doubt, on the stage at which it was performed—it is easily understood why a sublingual phlegmon, when the operation is performed early, might not have a localized pus cavity, but, if more time has elapsed before the operation, local abscess formation will have occurred.

Price[5] reported a case (the fourth of his series) in which pus was not found by deep external incision into the sublingual space; two days later, however, pus began to discharge from the wound.

4. Guerini, Vincenzo: Ludwig's Angina, Dental Brief **2**:28, 1906.

5. Price, J. W.: Ludwig's Angina: A Report of Four Cases Including One Autopsy, Ann. Surg. **48**:649, 1908.

In the case reported by Phillips,[6] pus was not found when the incision was made into the sublingual tissue; a few days afterward, however, pus discharged freely from the wound.

In Miller's [7] case, although only serum and dark blood escaped when the incision was made into the deeper structures under the tongue, four days later pus discharged freely from the wound.

Likewise, in Stetten's [8] case, when a blunt dissection was made into the sublingual space, pus was not obtained, but subsequently a free discharge of foul pus started.

Linon's case "produced no gas when the incision was made, but, on the following day, there was an escape of gas from the same incision."

Delorme opened the submaxillary gland in one of his cases and did not find any pus; he divided the mylohyoid muscle, and malodorous infectious pus escaped.

The fact that pus was not found in many cases in which an incision was made, either at the time of the operation or subsequently, does not substantiate the position of those who contend that in true Ludwig's angina there is a phlegmonous swelling without pus formation. In the presence of an enormous swelling about the neck, a small focus of pus situated deeply behind the muscles is not easily found. No doubt, many patients have developed more generalized evidences of infection and died before the local processes were revealed in the presence of pus formation. A review of the bacteria that have been reported as found will show that they are practically all organisms which form pus under certain conditions; and there are no conditions in these cases that would preclude the formation of pus when the organisms are introduced. To say that in any case of Ludwig's angina pus would be encountered, if an incision is made in the proper location, is not true. It is necessary to take into consideration the stage of the process at the time the incision is made. To say that there are certain cases of this disease in which pus formation does not occur, due to the type of the infecting organism, is begging the question. In substantiating this statement, it must be conceded that some cases which have all the appearances of a beginning Ludwig's angina are aborted without reaching the stage of pus formation. I recently had such a case in which, soon after a dentist had extracted a lower molar, a marked soreness appeared under the tongue. The patient could not bear to have the tongue pressed down on account

6. Phillips, A. E.: Notes of a Case of Angina Ludovici: Operation: Recovery, Lancet **2**:408, 1898.

7. Miller, H. T.: Ludwig's Angina: Report of a Case, J. A. M. A. **71**:1651 (Nov. 16) 1918.

8. Stetten, DeWitt: Ludwig's Angina: Case Presented, Ann. Surg. **81**:704, 1925.

of pain, and said it felt as if it were being pushed upward toward the roof of the mouth. Slight swelling appeared externally in the suprahyoid region. Vigorous treatment, instituted early, consisting of the thorough cleansing out the cavity from which the tooth was extracted and the constant application of ice packs externally and cracked ice internally, was followed in five days by the subsidence of all symptoms.

Any statement that could be made of Ludwig's angina in this connection could be made of infectious diseases elsewhere in the body. Beginning infections, whether general or local, are frequently aborted before they manifest the typical signs characteristic of their presence in the parts involved, whether it be in the abdomen, chest, head or any other section. To conclude that pus is not present because it is not encountered at the time of incision is to presume too much on the astuteness of the operator. Cases have been reported in which autopsy demonstrated the presence of pus which had been missed by incisions made prior to the death of the patient. In Ludwig's case there was copious suppuration, which ruptured into the mouth spontaneously.

DIAGNOSIS

The symptoms pathognomonic of Ludwig's angina are those referable to an infectious process under the tongue and confined within the floor of the mouth, namely, an elevation of the tongue and redness and edema of the mucous membrane over the area involved. When the infection is confined to one side, the tongue will be pushed to the opposite side. If it is bilateral, or near the midline, the tongue is pushed toward the roof of the mouth.

The redness and edema of the sublingual mucosa at times reach marked proportions. In one of my cases, it resembled a secondary tongue. It usually protrudes against the lower teeth and on examination will be found to bear their imprint.

The discomfort of the patient is marked from the beginning. Swallowing is painful. After the disease develops, speech becomes gutteral. There is noticeable dribbling of saliva, and respiration becomes embarrassed. Swallowing sometimes becomes impossible.

There is a boardlike swelling, externally, corresponding to the location of the disease inside the mouth. This swelling does not have any point of fluctuation, and, although it may be limited over a narrow space in the beginning, it usually spreads up and down the neck and envelops the throat, dog collar fashion. Pressure over the swollen area elicits marked tenderness.

The temperature varies from 99 to 103 F. Sometimes it goes as high as 104 F. In one of my cases the temperature remained below 100 F., while in another it went above 103 F.

There is a moderate leukocytosis, ranging from slightly above normal to 16,000.

The systemic reaction to the disease depends on the stage to which it has progressed. In the later stages, when secondary complications have set in, the symptoms will depend on the nature of those complications. Edema of the glottis and epiglottis are not infrequently encountered. When the infection has broken through the barrier at the posterior limitations of the sublingual space, it may become general sepsis, or a descending abscess of the neck, a condition ably discussed by Glogou.

PROGNOSIS

The greatest factor in the prognosis, in my opinion, is that of allowing the infection to break through the barrier at the posterior limitation of the sublingual space. If this occurs, there are two courses which it may pursue: It may be carried by the lymphatic glands to the structures in the carotid sheath, where it is prone to develop into a general septicemia; or it may become a descending abscess of the neck, following the muscle and fascia planes downward to the mediastinum.

Lockwood reported a case in which, at autopsy, he found a clot in the jugular vein on the involved side.

In Guerini's case, at autopsy, gangrenous and septic alterations were seen extending from the floor of the mouth as far as inside the thorax.

Another factor in the prognosis is the degree of immunity the patient has developed at the time he presents himself for treatment.

Waldapfel showed by experiments on himself that:

1. Streptococci cultivated from tonsils of a patient were destroyed by leukocytes of the latter to an extent which increased in direct ratio to the course of the convalescence.

2. This increase in resistance of the organism was directed specifically against the variety cultivated from the patient.

3. The streptococcus found in angina represented no occasional or secondary observation, but bore an intimate relation to the disease and its course.

TREATMENT

The treatment is thorough surgical drainage of the involved area. As previously indicated, this should be done early in the disease, before the infection breaks through its anatomic barriers. Except in those cases in which the abscess points near the surface inside the mouth, the incision of choice is external, cutting down over the location of the abscess, as indicated by the point of greatest swelling and tenderness. The incision should extend through the floor of the mouth, and should

be made long enough to admit two rubber tubes. If the abscess cavity is not entered at once, it is preferable to introduce forceps, closed, and then to open them out inside the floor of the mouth in different directions after the method of Hilton. When the abscess cavity has been evacuated, if there is pus, or when ample space for drainage has been provided in those cases in which pus has not yet formed, two moderate sized tubes should be inserted deep into the cavity and allowed to protrude from the surface. Through these tubes the cavity should be irrigated twice daily with some antiseptic solution, for example, 5 per cent mercurochrome-220 soluble.

It was stated in the anatomic description previously given in this paper that the location of the abscess had a relationship to the anatomic structures in the floor of the mouth. If the abscess forms lateral to the geniohyoid muscle, it will require a lateral incision, which will be necessary to divide only the mylohyoid muscle. However, if the abscess is in the midline, its location may be in either one of two places: above the geniohyoid muscle, between that muscle and the geniohyoglossus muscle or below the geniohyoid muscle. In the first case, the incision should be deep enough to divide both the mylohyoid and the geniohyoid muscles in the midline. In the second case, it would be necessary to divide only the mylohyoid muscle in the midline.

It will be necessary in many cases to give supportive treatment in the way of the Murphy drip method, etc., since the patients are unable to swallow food or water for some time.

CAUTION

It is not enough simply to make the proper incision. It is necessary to keep the incision open with rubber tubing, or by some other effective method. In the third case reported by me, the incision was packed with iodoform gauze. After ten hours of improvement following the operation, the drainage decreased and the symptoms grew worse. At the end of twenty-four hours, when the packing was removed, a considerable quantity of pus had collected in the cavity and could not drain. After the gauze packing was discarded for the rubber tubes, improvement was rapid.

Mikulicz advocated through and through drainage of the sublingual space. This, no doubt, is advisable in some extreme cases in which the quickest and most thorough drainage is imperative. Drainage into the mouth, however, should be avoided when justifiable, since there is danger of aspiration, or swallowing, of the infectious material, which might result in complications.

Rehn advocated extirpation of the submaxillary gland in difficult cases. Since the submaxillary gland is not involved in any uncompli-

cated case of Ludwig's angina, his operation does not come within the scope of this paper.

Intravenous medication, such as with mercurochrome-220 soluble and antistreptococcic serum, has been used by some with seemingly good results. In the early stages, before the time of pus formation, this procedure might be effective to abort the full development of the disease, as it does in infectious processes elsewhere. After the stage of pus formation has been reached, however, it is doubtful if this procedure has any effect on the course of the disease.

OSTEOMYELITIS OF THE FRONTAL BONE

REPORT OF FOUR CASES PRESENTING PROBLEMS IN ETIOLOGY AND TREATMENT *

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The certain prevention of a disease depends on knowledge of the specific cause. Thus a serious problem is encountered in combating osteomyelitis, the etiology of which is still a matter of dispute, although several plausible theories have been presented. In the long bones, where this disease has been fairly common for many years, trauma and hematogenous infection usually have been considered the causes. However, even here where demonstration is somewhat easier than in the frontal bone, I believe there is doubt in certain cases as to where the infective agent arose. Hastings [1] of Los Angeles reports a case of acute diffuse osteomyelitis of the frontal bone in which it would appear likely that the focus was of dental origin, and hence, hematogenous. Lynch of New Orleans, who is the originator, I believe, of the theory that osteomyelitis is caused by stripping up the periosteum and the subsequent trauma of the radical operation, makes out a strong case for his theory. However, he fails to account for the fact that some of the men who have been performing this operation for many years have not had any cases until recently, and others have had few at any time. I believe that the greatest cause of all, when osteomyelitis follows another operation, is failure to remove all necrotic material, which is thus pressed against denuded bone, the nutrition of which has been almost destroyed temporarily. The wound is deprived of air by the suturing of the soft parts over it, and the drainage is limited to the opening into the nose. Is it remarkable that reinfection should occur, and is it not a more reasonable cause of osteomyelitis than merely lifting up of the periosteum on the external frontal wall? In those cases in which the osteomyelitis is acute and not the sequel of another operation on the sinus, I believe the etiology lies in one or all three of these causes: (1) trauma, (2) long continued contact of infective material in an undrained cavity and (3) hemolytic bacteria, generally the streptococcus. All my cases have showed this type of infection, and all that I have personally investigated have shown it. It is within the past nine or ten years that this increase

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* Read at the Fiftieth Annual Congress of the American Laryngological Association, Washington, D. C., May 3, 1928.

1. Hastings, Hill: Acute Fulminating Osteomyelitis of Frontal Sinus; (?) Origin (Dental Focus): Report of a Case, Arch. Otolaryng. **6**:559 (Dec.) 1927.

in osteomyelitis of the frontal bone has been encountered, and this period coincides with that following the pandemic of so-called influenza, which sent thousands of carriers of the hemolytic streptococcus all over the world to disseminate this deadly organism. In a paper entitled, "A Scourge of Hemolytic Streptococcus," I [2] directed attention to the enormous increase in the number of cases of mastoiditis necessitating operation with this organism as the basic cause, which gave rise to the cases variously termed hemorrhagic, coalescent or hemolytic. Up to that time, such cases had been rare in my experience; they not only were no longer so, but actually became common. As I have said, I attributed this to the dissemination of the bacteria by the thousands of carriers who were everywhere. To the list of the possible causes, Hastings has added the hematogenous, and it must henceforth be included in the etiology, as his case is convincing.

These four cases present material for proving almost any or all of the various forms of etiology thus far offered, if one chooses to see his own theory in them. In fact, one of them alone could be used to show every one of the theories I have laid down, or that has been suggested by any one. Of course, tuberculosis and syphilis, the twin bane of all investigators in etiology, are not considered, as they are basic and are taken for granted. Moreover, they were not present in my cases. To open an osteomyelitic process and leave it wide open and drained until a sequestrum has formed and the delimiting process has been established cannot be questioned as a good surgical procedure in cases involving the long bones, but in the frontal bone, I think that the problem is somewhat different. Here the virulent infective material is left in contact with so poorly resistant a region as the cribriform plate of the ethmoid, and the thin inner frontal plate separates it from the brain. I lack the courage to use this procedure, even if I were convinced that it might help matters, and I am yet far from being so. I prefer to remove all softened bone and leave a wide open wound, though this might prove to be the same thing, as it is not possible to be sure that all diseased bone has been removed.

REPORT OF CASES

CASE 1.—A vigorous boy, aged 12 years, with a soft swelling to the right of the glabella 1 inch (2.5 cm.) in diameter and painful to the touch, presented himself to a colleague of mine. He seemed to think that it had followed an accidental blow about ten days before, and it was accordingly incised under the presumption that it was a periostitis with effusion. Free pus was obtained, two stitches taken and a small drain inserted in the lower angle of the wound. All symptoms seemed to have disappeared in ten days. Two months later, he came to me with the same symptoms, only this time the swelling was much more extensive; it was pear-shaped, and the narrow limb extended vertically to the

2. Dabney, Virginius: M. J. & Record **119:** (June 18) 1924.

hair line. The roentgenogram showed the absence of frontal sinuses, and outlined the collection of pus in conformity with the swelling. A vertical incision was made from the hair line to the supra-orbital ridge; a great quantity of pus was released, and a distinct deep channel sufficient to accommodate an ordinary pencil was found, which had been burrowed through the external frontal plate by the pus. There was, of course, no connection with the nose, as there was as yet no frontal sinus or even enlarged frontal cell. As no thought of osteomyelitis entered into my calculations, I merely smoothed the rough edges of the sinus and its bottom, sutured the upper two-thirds and inserted a gauze wick. Convalescence was uneventful, and a depressed scar resulted which hugged the contour of the pus sinus. Five years later, the boy returned with a history of frequent headaches, occasional swelling of the brow and nasal discharge, which had occurred frequently during the past two weeks, but since ten days before he presented himself swelling had become permanent. He had developed into a large, powerful young man with an unusually large head, and I found bad tonsils, a mucopurulent discharge from the right naris, swelling of the middle turbinate and a brow that was exquisitely painful on the lightest touch. The roentgenogram now showed small but fully developed cloudy frontal sinuses, complete absence of the vertical furrow of the previous operation and a dark right ethmoid sinus. His condition was critical; marked weakness, great pain, a septic complexion, temperature of 102 F. and chemosis of the right eye being noted. As the pus had extravasated high up on the frontal prominence, I first reopened the old vertical incision and joined this at right angles with the usual Kilian brow incision, realizing that the condition was osteomyelitis and that a cosmetic result was not to be considered. The furrow was completely filled with hard, healthy bone, the external plate over the right sinus was necrotic and soft, and it and half of that over the left sinus were removed, the bone imparting to the rongeur and curet the sensation that one gets in cutting a rind of cheese. The left sinus was opened through the septum, which was necrotic and showed a large erosion in the center. As the wound was kept wide open and there was no disease in the left ethmoid sinus, there was no necessity of disturbing the interior of the left naris. However, the right ethmoid sinus was completely exenterated. I have seen the patient at intervals of two years, and he still is free from recurrence, though I have since then removed his tonsils as a precaution. Several interesting features present themselves in this case, notably, the formation of new bone to fill a large cavity in the frontal bone, and the development of two frontal sinuses between the two operations. The two conditions were probably the result of the same process: the natural development of the bones in a boy between the ages of 12 and 17.

Case 2.—An obese, colored woman, aged 28, reported to the clinic at the Episcopal Hospital, giving as her chief complaint a painful swelling on her forehead of ten days' duration and a slight cold in the head. The cold referred to was seen to be a free discharge of pus from the right naris, apparently originating about the middle turbinate. A large fluctuating swelling about the size of a silver dollar was found 2 inches (5 cm.) above the right brow near the midline. The roentgenogram indicated pus in the right frontal and maxillary sinuses but a clear ethmoid and sphenoid sinus. It also outlined the frontal collection of pus. A Luc-Caldwell operation was performed on the antrum which was full of thick pus; a vertical incision was made to open the swelling above the brow, and this too showed free pus. The cavity here was thoroughly curetted, and the edges beveled to a point where the cavity was flush with the cortex, the bone coming away almost like stiff putty, and leaving a cavity 3 by 2 inches (5 by 7.6 cm.).

The usual Killian incision at the brow was made to join the vertical line, and a little pus was found in the frontal sinus, the external wall of which was softened just at the cortex above. The double wound was dressed as an open wound, but no opening was made in the ethmoid, as I believed the contamination of the frontal sinus was from the cortical abscess above and not from the antrum below. As the entire wound healed, with a healthy, depressed scar in a month, and a year has elapsed since that time, it would appear that the osteomyelitis has been arrested. The etiology here is not clear.

Case 3.—The first time I saw the patient, a woman, aged 55, her temperature was 101.5 F., her left eye was closed, and her left naris was completely blocked with pus and an enormous middle turbinate. However, there was no chemosis of the eye. Appropriate measures served to return the parts to normal in three days, with the exception of the middle turbinate, which receded somewhat but which was seen to be cystic. She had been forced to spend several months in Arizona for a number of winters because of what she said was "sinus trouble." Two months later, in accordance with clinical and roentgenographic evidence of a pansinusitis, I tried to persuade her to have a Killian operation performed to be followed by a Luc-Caldwell operation, but all I accomplished was permission to make an exenteration of the ethmoid and sphenoid sinuses under local anesthesia, followed after two months by a Luc-Caldwell operation; all of these procedures were bilateral. During these operations I found only what is usually found in any neglected pansinus conditions. A year later, I performed a conservative Killian operation on the left side, and found in the frontal sinus only a small encapsulated collection of pus just over the nasofrontal duct; the remains of the ethmoid sinus were small and were removed at this time. The interfrontal septum was healthy and the cortex and internal frontal plate hard; these points are important in the light of the subsequent developments. A month after healing, the wound suddenly swelled and the eye became closed almost over night. I at once reopened the wound, and this time found the frontal sinus partly filled with degenerated tissue. There was also some necrosis in the middle ethmoid sinus, which I had overlooked or which had occurred after the other operation. The wound was closed, and I had to reopen it five weeks later. This time, however, I recognized the condition, and I removed every vestige of necrotic material, including the supraorbital ridge, the entire floor of the sinus and all the bony postocular support. The interfrontal septum was roughened, and on curettement showed an erosion into the other sinus. This was enlarged, and a mucopurulent discharge trickled out, but the patient's condition was too critical to admit of an operation on the sinus. At present, after nine months, there are no symptoms referable to the unoperated sinus, and the wound has healed solid but is depressed, as I carried the curettement far up on the cortex and beveled all edges. The history of the case (at least five years), the short time between operations (about a month) and the consistent hardness of the cortex until recently suggest an osteomyelitis from the beginning, mild but persistent, increasing in virulence until it became too manifest for me to overlook. I feel sure that long contact with infectious material was the cause. Of course, this time I left the wound wide open.

Case 4.—A woman, aged 21, Dr. Joseph Bryan's patient, came to me after he had performed several thorough and careful operations, but was forced to relinquish control of the case as he had to leave the city for some time. In November, 1925, the patient reported to him with the history of a severely swollen and painful right eye for the preceding three months. The roentgenogram and the report on the eye indicated the necessity for a radical operation on the nasal accessory

sinuses of the right side, which was accordingly performed by the technic which Dr. Bryan originated, and which is similar to the Killian technic in its essential points. In accordance with the symptoms and clinical indications, the following operations were found necessary and were performed by Dr. Bryan: Jan. 28, 1926, operation on the intranasal ethmoid and sphenoid sinuses under local anesthesia; February 11, reopening of the frontal sinus and the rest of the wound; June 26, complete radical operation; Feb. 9, 1927, operation on the ethmoid and sphenoid sinuses under local anesthesia, intranasal; February 21, complete radical operation (fourth radical operation). It was at this last operation, at which I was assisting, that I came into contact with the case. While the disease was extensive with a burrowing into the glabella, I did not suspect osteomyelitis nor do I think that Dr. Bryan did. A few days later, I took charge of the case, and on May 1, reopened the old wound and found it necessary to remove the interfrontal septum, all the cortex over the right sinus and half of that over the left and the entire floor of the left sinus. The glabella was so soft that it came away under the curets like the shavings of a soft pine board under the plane. The necrotic process had attacked the cortex to a distance of 3 inches (7.6 cm.) above the supra-orbital ridge. The left ethmoid and posterior and lateral walls of the orbit were all cleaned carefully. The old wound on the right showed an erosion in the center of the internal frontal plate, through which a bubble of dura bulged. The wound was left wide open, but as the patient was septic after two years' infection, it was slow to heal, which it did in two months. From week to week, some small area would break down and under local anesthesia would yield to curettement. The patient's progressive weakness and the tendency of small spots to reopen led me to have the hemoglobin content tested; on finding it only 44 per cent, I learned that she had had a bad menorrhagia for six weeks. She was put in the hospital, given two transfusions of whole blood into the vein, two into the buttocks and two applications of radium in the cervix. These measures resulted in an immediate improvement, and she is now home and the osteomyelitis apparently cured after eleven months. This case, I think, shows clearly that long contact with diseased material produced the osteomyelitis, as four thorough operations showed the same pathologic process; but it was only on the fifth operation with an open dressing that the arrest of the process took place. I believe the condition was osteomyelitis from the first operation, though there was no reason to suspect this from the gross appearance. In the future, when I get a definite history of long-standing disease of the sinuses and find marked and extensive destruction associated with a hemolytic organism, I shall leave the wound at least partly, if not wholly, open. I believe every one of these four cases was of osteomyelitis origin from the beginning, but was not recognized. It is striking that despite the most thorough, painstaking and radical surgical procedures, none of the wounds healed solidly until the open dressing was adopted, the number of operations varying from three to five, not counting those performed intranasally, before the disease process could be arrested. As anemia is always one of the striking and invariable complications of osteomyelitis, especially when a hemolytic organism is found, this should be corrected by transfusion. The healing of a wound is, of course, a metabolic process, and it is seriously interfered with when anemia is present.

CONCLUSIONS

Osteomyelitis of the frontal bone following radical surgical procedures is due, not to the operation and its technic, I believe, but to long contact with undrained infective material. Otherwise, why have many

rhinologists had no such cases even though they have been performing this operation for many years?

I believe another potent cause are the hemolytic bacteria, especially the streptococcus, which may account for the increase in these cases, coinciding with the dissemination of this germ since the World War through carriers who have returned to their homes all over the world.

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THE EFFECT OF OTITIS MEDIA ON PATIENTS PREVIOUSLY OPERATED ON FOR MASTOIDITIS *

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A second attack of otitis media may occur long after the initial attack during which a mastoid operation may have been performed. The idea is prevalent that if a patient has been operated on for acute mastoiditis subsequent infection is not likely to occur. There really is little foundation for such a view.

As a matter of fact, certain persons seem predisposed to involvement of the middle ear with acute infection of the upper respiratory tract. It is not likely that the infecting organism will show any particular selectivity for the ear. It is more likely that the particular anatomic configuration of the nasopharynx and eustachian tubes, together with the person's habits with regard to the hygiene of the infected upper respiratory tract, are the important factors involved. An example of this is the habit of blowing the nose in a wrong manner.

Animals are seldom affected with otitis media, principally, in my opinion, because they are not able to clear out the nose as the human being does. They sneeze or snuff in the natural way and thus clear the nose and nasopharynx. Man, in his ingenious way, has attempted to improve on this natural physiologic mechanism. He has found that by holding one nostril closed (always the physiologically open side), he can blow against the closed side and clear the nose effectively if the blockage is due to secretion. If, however, the blockage is due to swelling of the membrane before any secretion has collected, such blowing causes a strong positive pressure in the nasopharynx, resulting all too frequently in strenuous auto-inflation of the middle ear. The swelling of the mucous membrane within the tube is not able to resist this, and infection is carried to the middle ear. Many patients say that they first noticed discomfort within the ear after blowing the nose in this manner.

Infection of the middle ear may occur, however, in cases in which this manner of blowing the nose has not been practiced. A cursory search of the literature, including textbooks, for suggestions as to the care of reinfected ears has been fruitless. Perhaps surgeons have felt that the subsequent infection is a reflection on them, but this should not be the case.

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* From the Section on Otolaryngology and Rhinology, The Mayo Clinic.

* Read at the Meeting of the American Laryngological, Rhinological and Otological Society, Washington, D. C., May 3-5, 1928.

Reinfection of the middle ear and the mastoid wound occasionally occurs. It has been my experience that if the original operation on the mastoid has been complete, there need be little concern. If all the mastoid cells have not been opened at the time of the original so-called simple mastoid operation, further surgical intervention may be indicated. By way of digression, let me say that the term "simple mastoid operation" is poor, for it may mean just what it implies both from the standpoint of the patient who may feel that it is of little significance or that it is not serious, and from the standpoint of the inexperienced surgeon who may think it is of little moment. I do not believe life has ever been saved by a simple drainage operation, whereas many lives have been saved by complete removal of the infected mastoid cells. For this reason, I prefer to call the operation "the complete mastoid operation."

Fifty-three cases of subsequent reinfection of the ear and mastoid have been encountered in which the so-called simple mastoid operation had been performed elsewhere. In all but three cases, the severity of the symptoms and signs made it necessary to reoperate. It was found in all cases that the mastoid cells had not been operated on completely. The tympanic antrum had reformed as a space in the scar tissue in those cases in which the cells had been removed from around the antrum. It seemed apparent that after the drum had healed, the physiologic ventilation of the ear had forced the contraction of the scar.

In two cases in which I had performed a complete mastoid operation previously, the mastoid scar suppurated, and simple drainage, similar to that which would be used for any superficial suppuration, effected rapid cure. Three cases of acute otitis media without symptoms or signs of mastoid disease ran the usual course after myringotomy.

SUMMARY

Reinfection of the middle ear and mastoid may occur. It is not a reflection on the surgeon. If a complete mastoid operation is performed, the safety of the patient is practically assured. If simple drainage only is carried out, more complete surgical intervention may be necessary in case of subsequent infection. Following practically all operations for acute mastoiditis, the tympanic antrum is reformed in the scar. Myringotomy and drainage of the mastoid scar after suppuration will effect a rapid cure if the operation on the mastoid process has been complete.

Clinical Notes

LARYNGEAL NECROSIS AS A POSSIBLE COMPLICATION OF THE TREATMENT OF GOITER BY IRRADIATION*

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It is more or less of a therapeutic axiom that in the use of any remedial agent, one should consider not only the primary effect desired, but the possibility of untoward by-effects or after-effects.

This axiom is particularly applicable to the use of the roentgen ray, the effect of which on tissue has been extensively studied, and which, under varying combinations of circumstances, has been shown by both experimental and practical experience to be capable of doing as much harm as good.

The average person is prone to think of damage to the tissue by the roentgen ray as either an acute burn from intensive exposure or as a malignant degeneration following prolonged or repeated exposure. Recent studies have shown the possibility of another type of lesion, essentially trophic in type, of particular importance because its manifestations may be delayed.

The primary effect of the roentgen ray on tissue is stimulation followed, when prolonged, frequently repeated or repeated over long periods of time, by varying degenerative reactions which have been studied and reported by Aschoff,[1] who regarded the trophic reactions following prolonged or intensive irradiation as due to changes in the endothelium leading to obliteration of blood vessels, and Lowen,[2] who commented that, because of injury of the blood forming tissues resistance to bacterial attack is decreased.

In a recent paper, Iglauer[3] reported one case of his own and twenty collected from the literature in which severe laryngeal injury followed roentgen-ray treatment.

Clerf[4] added reports of six more cases, several of which are of interest because of the long interval between the treatment and the after-effect, a fact which is the genesis of the present report, which is believed to add one more case to those already recorded.

In one of Clerf's cases, similar in many respects to the case reported here, in which the roentgen ray had been applied for the treatment of a patient with exophthalmic goiter, laryngeal symptoms appeared five years after the last treatment.

At autopsy, the larynx was a putrid, gangrenous mass. In another case, also of goiter, the last treatment was given nine years prior to the development of trophic changes leading to necrosis of the laryngeal cartilages.

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*From the Laboratories of the Atlantic City Hospital.

1. Aschoff, L.: Pathologische Anatomie, ed. 6, Jena, G. Fischer, 1923, p. 82.

2. Lowen: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **19**:141, 1908; quoted by Wells, H. G.: Chemical Pathology, ed 5, Philadelphia, W. B. Saunders Company, 1925, p. 421.

3. Iglauer, S.: Roentgen-Ray Injury to the Larynx, with Report of a Case, Ann. Otol. Rhinol. & Laryngol. **36**:136, 1927.

4. Clerf, L. H.: Laryngeal Complications of Irradiation, Arch. Otolaryng. **6**:338 (Oct.) 1927.

In none of the cases reported by Clerf was there any systemic disease, recent infection or local injury to account for the laryngeal lesions which the author was convinced were due to prolonged and repeated roentgen-ray exposures. He believed, therefore, that in view of the possible untoward effects, roentgen-ray exposure of the neck is a source of potential danger until more can be learned of individual susceptibility and cumulative effects.

In view of the following case report, these conclusions seem deserving of serious consideration.

REPORT OF CASE

S. B., a white woman, married, aged 32, was first seen in October, 1925, by Dr. H. S. Davidson, through whose courtesy the present report is possible.

At that time, she presented a history of having been treated by roentgen-ray exposures for exophthalmic goiter "for several years." Details as to the frequency and duration of the treatments were not elicited at this time and have not since been obtained, but the assumption seems warranted that the patient had received a number of such treatments extending over a long period of time, the last being administered in 1923. The results were satisfactory, the basal metabolism being reduced from plus 53 to normal.

On Sept. 16, 1927, the patient consulted Dr. Davidson for a minor ailment. The basal metabolism on that date was minus 8, and the blood count was as follows: hemoglobin, 11.73 Gm. per hundred cubic centimeters (85 per cent); red blood cells, 4,910,000; white blood cells, 14,400; color index, 0.8; differential count: polymorphonuclears, 73 per cent; large mononuclears, 2 per cent; small mononuclears, 24 per cent; eosinophils, 1 per cent. Abnormalities were not seen in a study of the stained smear.

On Nov. 12, 1927, the patient was seen by Dr. Davidson because of headache and soreness in the neck and throat. She had a temperature of 101 F. On the following day, hoarseness and perceptible difficulty in swallowing were present. Smears and cultures from the throat were negative for diphtheria and Vincent's angina. The temperature remained elevated, however; the hoarseness and dysphagia persisted, and the patient was evidently prostrated and toxic.

A blood count at this time showed a total leukocyte count of 1,100 per cubic millimeter, with 40 per cent polymorphonuclears, 52 per cent small lymphocytes, and 8 per cent large lymphocytes.

An examination by a nose and throat consultant gave negative results, but as the patient's condition continued to get worse a second consultant was called in who found gangrene of the arytenoid cartilages. As the patient was being prepared for tracheotomy, she died.

A limited autopsy was performed within an hour after death. The skin on the anterior portion of the neck in the region of the thyroid and larynx and for some distance on either side was deeply bronzed, which resulted in a sharply demarcated band approximately from 2 to 3 inches (5 to 7.6 cm.) wide. As the skin flaps were reflected in this area, there was a slight but perceptible edema of the superficial underlying tissues. On exposure of the thyroid, this organ, and especially the transverse isthmus, was shrunken and thinned out. Gross obstruction of the trachea below the larynx was not found. When the larynx was opened a greenish-black area of gangrene involving the vocal cords and adjacent tissues and the contigous portion of the base of the tongue was exposed. The tissues had a foul odor.

Cultures and smears from the gangrenous area showed a mixed and varied bacterial flora in which streptococci and pneumococci were numerically predominant. The bacteriologic studies did not reveal any evidence of diphtheria or Vincent's angina, and did not suggest a bacteriologic etiology for the gangrenous process.

Histologic examination of the laryngeal tissues showed a diffuse gangrenous process which, in the main, involved only the superficial tissues.

In the absence of any evident or demonstrable cause for the gangrenous process, the patient's past history was investigated, and the fact of the treatment of the goiter by roentgen ray was elicited.

The deep bronzing of the skin of the neck and the shrunken thyroid with adjacent adhesions both suggested repeated if not prolonged exposures. It was thought, therefore, that while it was impossible from the evidence at hand to evaluate correctly the effect of the exposures on the tissues or their relation, if any, to the terminal lesion, devitalization of tissue was at least a contributing factor in the production of the terminal process.

The fact that the patient had not been exposed to the roentgen ray for a period of five years preceding the final illness casts doubt on this assumption. However, Clerf's report was later encountered, and his two cases of a similar condition, one occurring five and one nine years after treatment, revived the possibility and suggested the publication of this report for others to corroborate or controvert by future experience and observation.

LAVAGE OF THE MAXILLARY ANTRUM THROUGH THE NATURAL ORIFICE IN INFANTS*

SIDNEY YANKAUER, M.D., AND IRVING B. GOLDMAN, M.D., NEW YORK

The interest in disease of the sinuses in infants and children prompted us to attempt the investigation of the maxillary antrum. For this purpose, we have successfully employed a small cannula (fig. 1) which enters the natural orifice of the cavity in infants. The cannula is 3 inches (7.6 cm.) long and is of a small bore. At the distal end it is curved at right angles to the shaft. The curved arm is approximately three-eighths of an inch (0.9 cm.) in length. The cannula is a miniature of the one devised for adults by Dr. Sidney Yankauer.

Although the antrum in an infant is small, the natural opening is relatively large. The hiatus semilunaris can be seen in the infant cadaver as a rather broad gutter, at the posterior extremity of which is found the ostium of the antrum. Because of the flexibility of the soft structures of the middle meatus, the cannula tends to be guided toward the orifice when it reaches the hiatus semilunaris. The requisites for success in this procedure are the knowledge of the location of the ostium and the employment of the cannula as a probe.

PROCEDURE

For the purpose of demonstration, the head of an infant cadaver was divided into two parts. Both nasal chambers were then brought into view by the excision of the septum (fig. 2). On the left side, the middle turbinal was removed. The cannula which will enter the natural orifice was then passed along the upper surface of the inferior turbinal with the tip facing the lateral nasal wall. As the hiatus semilunaris was approached, the tip was rotated outward and downward, and was directed into the natural orifice. On the right side (fig. 3), with all structures present, the cannula was passed in a similar

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* From the Rhino-Laryngological Service of the Mount Sinai Hospital.

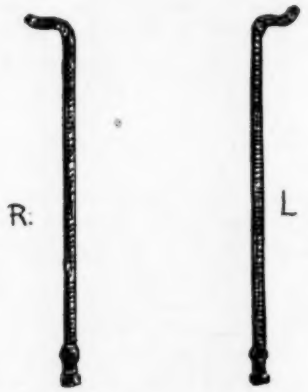

Fig. 1.—Cannulas, right (*R*) and left (*L*), used in natural orifice.

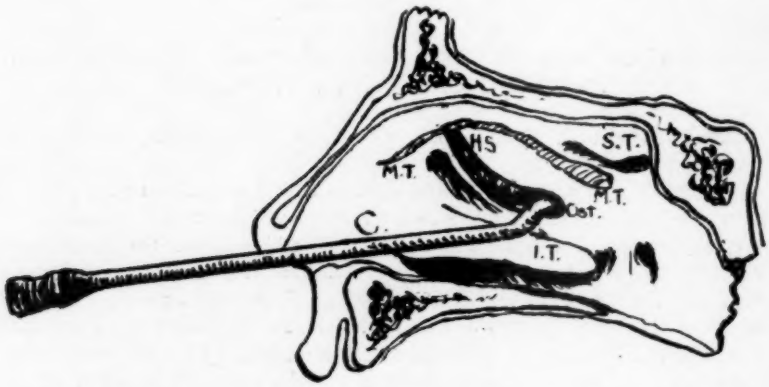

Fig. 2.—Cannula used in natural orifice in situ; middle turbinal has been removed. *C* indicates cannula; *M.T.*, middle turbinal; *H.S.*, hiatus semilunaris; *I.T.*, inferior turbinal; *Ost.*, ostium; *S.T.*, superior turbinal.

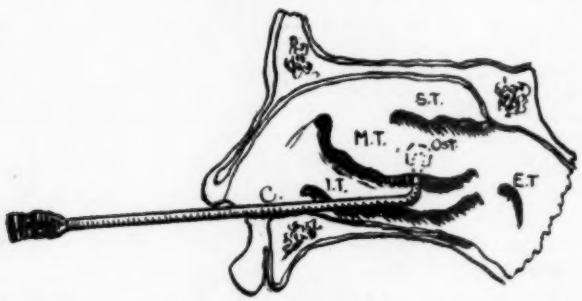

Fig. 3.—Cannula used in natural orifice in situ. *C.* indicates cannula; *I.T.*, inferior turbinal; *M.T.*, middle turbinal; *S.T.*, superior turbinal; *Ost.*, ostium; *E.T.*, eustachian tube.

manner along the inferior turbinal and it again entered the natural orifice of the maxillary antrum.

In an intact head of an infant cadaver, with the aid of a nasal speculum and the reflected light of a head mirror, the cannula was introduced into the orifices of both antrums. These were entered as was evidenced by the sensation imparted to the fingers during the procedure. Roentgenograms showed the

Fig. 4.—Anterior posterior view of cannula used in natural orifice in right maxillary antrum of cadaver.

cannula within the antrum (figs. 4 and 5). Similar results were obtained in a number of other specimens.

In the living infant, we found it necessary to use epinephrine hydrochloride solution in order to shrink the mucous membrane of the middle meatus. After shrinkage, the cannula was inserted and the orifice was entered. Entrance into the antrums of living infants of different ages has thus been possible. The

following tabulation conveys an idea of the various ages of infants whose antrums were entered through the natural ostia.

6 weeks	3 months	14 months
10 weeks	5 months	11 months
14 weeks	6 months	21 months
		24 months

CONCLUSIONS

This method is advocated because:

1. It is feasible and can be easily carried out in infants and children.

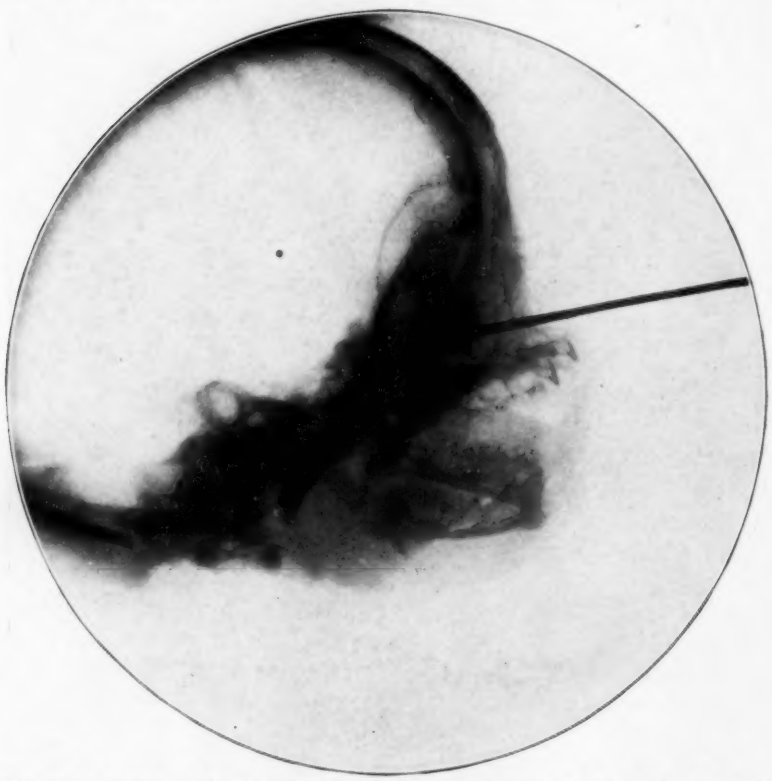

Fig. 5.—Lateral view of cannula used in natural orifice in right maxillary antrum of cadaver.

2. The necessity for antral puncture and the dangers which attend it are thus avoided.

3. The use of a general anesthetic for antral irrigation in infants is eliminated.

4. It can be employed as a diagnostic measure, especially in the search for focal infection in cases of gastro-intestinal intoxication and nutritional disturbances in infants.

5. It can be utilized as a therapeutic measure.

6. By the use of the cannula[1] which will enter the natural orifice, the study and investigation of antral disease in infants will be greatly facilitated.

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1. These devices are manufactured by Pilling & Sons Company, Philadelphia.

NEW HEAD-REST FOR ENDOSCOPIC EXAMINATION*

HENRY M. GOODYEAR, M.D., CINCINNATI

The head-rest which I wish to present was designed by Dr. Hasslinger in Hajeck's Clinic in Vienna. While working in this clinic, I was much impressed with the advantages of this ingenious device and brought one home for further study (fig. 1).

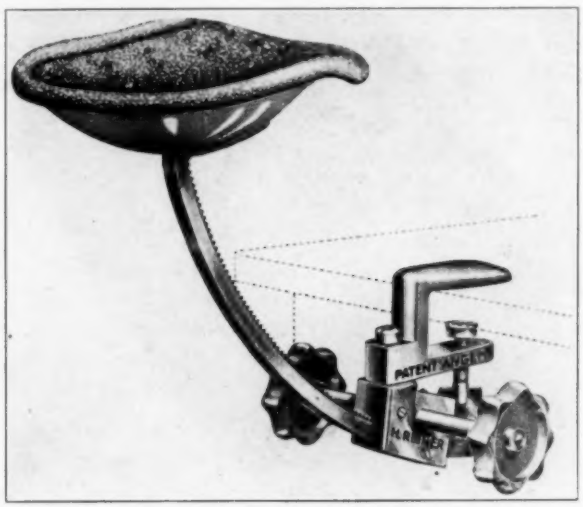
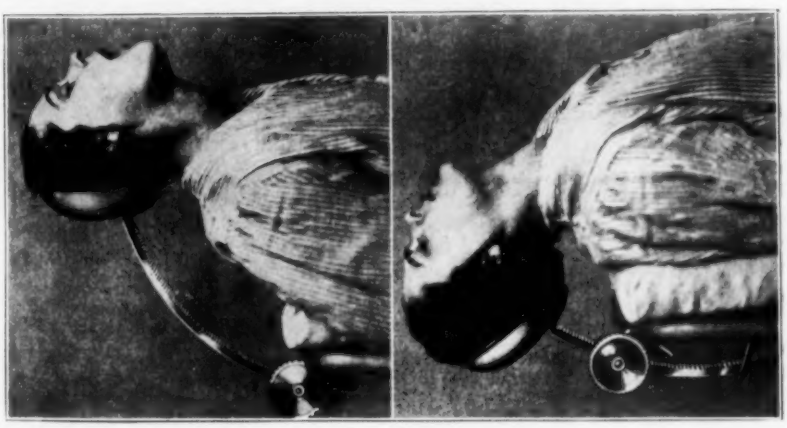


Fig. 1.—New head-rest.



A B

Fig. 2.—Head-rest in two positions.

When one depends on an assistant to hold the head in a uniform position by use of the hand alone, the head is often unsteady and the soft parts of the

* Shown before the Cincinnati Oto-Laryngological Society, March 13, 1928.

neck unrelaxed. Again, it may be impossible to obtain the services of a trained assistant, in which case the operator may bring the head in any desired position for a prolonged period by means of the movable head-rest.

With this new device, it is not only possible to retain the head and cervical spine in marked dorsoflexion, but the elevation can be changed to any desired position by means of a small wheel which locks automatically (fig. 2).

The head-rest, being fixed on a curved arm (fig. 3), describes an arc following the natural movements of the head, which is a distinct advantage over an elevation made in the direct vertical plane. All lateral movements are easily obtained, as the body of the entire mechanism swings free on a vertical axis.

556 Doctors Building.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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INTRADURAL DISEASES IN RELATION TO RHINOLOGY AND OTOLOGY

A CRITICAL SURVEY OF THE RECENT LITERATURE *

WELLS P. EAGLETON, M.D.
NEWARK, N. J.

I. ADVANCES IN THE KNOWLEDGE OF THE PHYSIOLOGY OF THE CEREBROSPINAL FLUID TENSION

Future advances in the surgical treatment of suppurative diseases of the brain will depend largely on the utilization of the physiologic factors influencing the central nervous system.

A. NORMAL VARIATIONS IN THE FORMATION OF CEREBROSPINAL FLUID AT DIFFERENT HOURS OF THE DAY

Hart [1] has demonstrated clinically that there is a marked variation in the formation of cerebrospinal fluid for the different periods of the day and night, there being a decrease in the early morning and afternoon, an increase in the forenoon and evening, with cessation in the formation of the fluid for the first few hours after midnight. He thinks that the variations in the amount of fluid formed is probably related to general vital activity, and possibly parallels the blood pressure curve. Air, blood, etc., should be classed as irritants which upset the normal fluid variations.

B. RELATIONSHIP BETWEEN CAPILLARY BLOOD PRESSURE AND THE INTRA-OCULAR TENSION

It is now generally recognized that the cerebrospinal fluid is a dialysate as is also the aqueous humor of the eye. In France, an effort has been made to measure the degree of the intracranial pressure by studying the amount of pressure necessary to produce a venous pulse at the fundus, the contention being that with the increased degree of pressure necessary to produce a pulsation in the retinal veins, there is also an increased intracranial pressure, consequently that the venous intraocular pressure furnishes a guide to the degree of intracranial pressure.

* In this survey I desire to acknowledge the assistance rendered by Dr. F. Robbins, New York, who has furnished the majority of the translations. Without her aid, this year's work would not have been possible.

1. Hart, D.: Cerebrospinal Fluid: Normal Variations in the Rate of Formation, Arch. Surg. **15**:943 (Dec.) 1927.

Although I tried the method on many occasions, cumbersomeness of this instrument has prevented any practical result. Lambert and Silbert's [2] experiments now offer a guide to the measurement of the intra-ocular capillary blood pressure which may be of sure value in measuring the intracerebral capillary pressure, as both intra-ocular, aqueous and the intracranial cerebrospinal fluids are dialysates, from the blood; although they do not suggest it I think that by this means one may be able to arrive at the degree of tension of the brain bulk, and thus the intracranial pressure. Lambert and Silbert report the effect in intra-ocular pressure of intravenous injections of hypertonic salt solution. They state that the main factors controlling the secretion-absorption equilibrium of these fluids are the hydrostatic pressure of the blood (capillary blood pressure), the osmotic pressure of the blood and the capillary permeability. After the intravenous injection of a hypertonic 5 per cent solution of sodium chloride, they found that within forty minutes the intra-ocular tension fell 40 per cent if 300 cc. was injected. If 150 cc. was used, the fall was 21 per cent. The tension of the eyeball returned to normal in from one to two hours. They are of the opinion that the fall in pressure is of a similar nature to that which occurs in advanced diabetes.

I wish to call attention to the fact that ligation of the common carotid, in septic diseases at least, causes a fall of the intra-ocular tension of the homolateral side of about 50 per cent, and that this decreased intra-ocular tension continues for several days.[3]

C. THE DEGREE OF THE CEREBROSPINAL FLUID TENSION A MEASURE OF THE DEEP VENOUS PRESSURE AND OF THE BLOOD MASS

On the other hand, Tzanck and Renault [4] believe that the degree of the pressure of the cerebrospinal fluid is a measure of the degree of the deep venous pressure, both being altered by the blood mass. They found that in man the withdrawal of 200 Gm. of blood lowered the spinal pressure 6 cc. of water. They think that hypertensions of nervous or meningeal origin exert no important effect on the venous pressure. On the contrary, the deep venous pressure is closely related to the pressure of the cerebrospinal fluid. These two tensions, cerebrospinal and deep venous, are, in their opinion, dependent on the same factors. Phenomena modi-

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2. Lambert and Silbert: Effect of Intra-Ocular Pressure of Injections of Hypertonic Salt Solution, J. A. M. A. **90**:1435 (May 5) 1928.

3. Eagleton, W. P.: Otitic Meningitis, J. A. M. A. **87**:1544 (Nov. 6) 1926.

4. Tzanck, A., and Renault, P.: Spinal Fluid Hypertensions of Mechanical Origin (Cerebrospinal Fluid and Blood Mass), Bull. et mém. Soc. méd. d. hôp. de Paris **43**:1441, 1927; Spinal Fluid Tension and Deep Venous Pressure, ibid., p. 1444.

fying the spinal tension (compression of the jugular veins, abdominal pressure, coughing, straining, etc.), act through the blood mass by causing a local or generalized deep venous hypertension. The cerebrospinal fluid is enclosed with the venous plexuses in the bony vertebral canal. When the venous system in this cavity becomes dilated, a considerable spinal hypertension is immediately observed; on the contrary, any hypertension from local causes in the cerebrospinal fluid will act but slightly on the deep venous pressure, because of the multiple anastomoses of the venous system and also because of the small volume of the cerebrospinal fluid as compared to the blood mass. The spinal fluid tension indicates the changes in the pressure of the deep venous circulation.

D. OCULO-AURICULAR MOVEMENTS OF ASSOCIATION

An associated muscular movement of the eyes and ears was discovered by Wilson [5] in 1908. He found that when the eyes are deviated extremely to one side there is a slow contraction of one of the intrinsic muscles of the homolateral ear, the musculus transversus auris. Brickner [6] examined 200 persons in relation to this oculo-aural phenomenon. He found that the oculo-aural response is present in cases of paralysis agitans, and therefore, he thinks it is unlikely that the reaction is one of the automatic associated movements supposedly mediated through the corpus striatum and the substantia nigra. There is a striking diminution in the range of the oculo-auricular movement in the presence of even a slight weakness of the facial nerve, consequently, he suggests the possibility of using this associated muscular phenomenon in determining the pressure or absence of doubtful lesions of the facial nerve.

II. PATHS OF INFECTION INTO THE MENINGES FROM LATENT INFECTIONS IN THE NOSE AND EAR

A. RECRUDESCENCE OF LATENT INFECTIONS IN SINUS DISEASE FOLLOWING AN OPERATIVE TRAUMA

Rosenow's [7] case of thrombosis of the cerebellar and veretebral arteries associated with intermittent hiccup is of especial importance to the otologist as it illustrates the symptoms from the thrombosis of the posterior inferior cerebellar artery and its relation to the production of nystagmus and hiccup. In addition, the case is of the greatest surgical

5. Wilson, S. A. K.: Rev. Neurol. & Psychiat. **6**:331, 1908.

6. Brickner, Richard M.: The Oculo-Auricular Associated Movement (Its Possible Utility as a Test of the Function of the Facial Nerve), Arch. Neurol. & Psychiat. **19**:104 (Jan.) 1928.

7. Rosenow, E. C.: Thrombosis of the Cerebellar and Vertebral Arteries Associated with Intermittent Hiccup, Arch. Neurol. & Psychiat. **18**:348-356 (Sept.) 1927.

importance because of Rosenow's careful experimental work which demonstrates that trauma inflicted on a patient who is the host of a quiet lesion, such as a nasal sinus or bladder infection, can light up a blood stream infection, which, if the micro-organism has a selective action on the cerebral spinal system, may cause grave infections in the central nervous system.

It has been the experience of many that operations—selective operations, such as for the cure of chronic suppuration in frontal sinus disease—have resulted in meningitis, and this when there was no disturbance of the internal wall of the sinus. There are a large number of such cases on record, and it is a real danger, as I can testify, as I have compiled five such cases which have occurred in one hospital with which I am connected. A London bacteriologist will not allow the immediate extraction of a tooth which has an infected area at its apex until the dental surgeon has first "rocked the tooth" in order that at first only a limited amount of the bacteria may be thrown into the system. By this small amount of infection immunity is established which prevents a severe infection when the tooth is extracted and a large dose of micro-organisms thrown into circulation.

History of Rosenow's Case.—Immediately after a cystoscopic examination on January 19, the patient had an increase of temperature for four days, which became normal for three days, but on January 27, it again rose to 103 F. He was unable to talk above a whisper and had difficulty in swallowing. The next morning he had intermittent attacks of hiccups. He vomited and had several sinking spells in which he became extremely cyanotic. The swallowing, choking and syncopal attacks grew rapidly worse, and paralysis of the right leg and arm ensued.

Examination revealed slight horizontal nystagmus, loss of left corneal reflex, crossed anesthesia of the left side of the face and the right side of the body—all symptoms of left vagohypoglossal pharyngeal paralysis. The neurologic signs thus indicated a lesion on the left side of the upper medulla involving the nucleus of the ninth, tenth, eleventh and twelfth cranial nerves and the spinal root of the fifth nerve. The patient died suddenly in one of his choking attacks two days after the onset of symptoms.

Autopsy revealed multiple septic foci from a chronic sinusitis with nasal polypi and hypertrophy of the prostate with acute and chronic cystitis and ureteritis and pyelonephritis. There were multiple abscesses in the kidney, edema of the lungs, with marked arteriosclerosis, especially of the cerebral vessels, and a retrograde thrombosis of the left posterior inferior cerebellar and cerebral arteries, with a small aneurysm of the vertebral artery at one point.

Sections of the brain showed areas of degeneration. Most of the small vessels in the area of the medulla in the region of the olive (that is about the level of the nucleus of the vagus nerve), were thrombosed. Around the thrombus there was a variable amount of round cells and most of the blood vessels were surrounded by edema and polymorphonuclear infiltration. A search for bacteria revealed a moderate number of gram-positive diplococci, singly, in small groups or in chains, within and adjacent to the region in the medulla and the infiltrated sphere over the left aspect of the medulla and within the thrombus in one vessel.

During the life of the patient, cultures and inoculations in animals were made from the secretions of the nasopharynx and from the urine, and also after death from emulsions of the brain and from blood aspirated from the heart and brachial veins.

Cultures from the blood of the patient yielded a large number of green producing colonies of streptococci together with a few colonies of *Bacillus coli.*

Experimental Injections of Micro-Organism Obtained from Secretions of Nasopharynx, Blood and Urine of Patient.—The injections into two rabbits of the swabbings from the nasopharynx produced in the animals tremors and twitchings of the various muscles, beginning in the masseter and spreading to the muscles of the ear, neck, shoulders and abdomen. Coincident with the appearance of spasms of the muscles, there was a horizontal nystagmus, difficulty in breathing and rhythmic spasms of the diaphragm.

One of the rabbits so inoculated, recovered; the other died. The necropsy of the rabbit showed congestion of the vessels of the meninges over the medulla and spinal cord and a turbid cerebrospinal fluid. Cultures from the brain of the rabbit yielded colonies of the streptococci—those from the blood gave no growth but yielded the same streptococci in dextrose from the brain broth cultures.

Cultures from the brain of the patient and from blood aspirated from the heart and brachial vein after death yielded a pure growth of streptococcus. Inoculation of a rabbit from these cultures was followed by respiratory difficulty and paralysis of the four extremities. Postmortem examination of the inoculated rabbit gave a marked congestion of the vessels of the meninges, a large hemorrhage at the base of the brain near the point of exit of the optic nerve and over the right lateral aspect of medulla, and hemorrhagic edema over the anterior part of the medulla, upper cervical cord and surrounding cervical nerve roots.

The streptococcus from the urine of the patient was injected into two rabbits. Spasms of the diaphragm occurred; they had marked twitchings and tremors of the masseters and muscles of the ear, neck and abdomen, diaphragm and shoulders. Cultures from the brain of the rabbit yielded streptococci; those from the blood showed a few colonies.

Sections of the animal's brain that had been injected with the streptococcus from the nasopharynx and the blood and the urine of the patient, showed a localized necrosis and leukocytic infiltration in the right frontal lobe at the point of inoculation, as well as leukocytes. There was infiltration in the lateral ventricle surrounding the choroid plexis.

Of course, Rosenow has long contended that certain strains of bacteria have a selective action on certain tissues, and in my opinion, the foregoing naturally helps to establish the truth of this contention.

B. MICROSCOPIC PATHS OF INFECTION

(a) From Nose by Way of Arachnoid Prolongations Surrounding the Olfactory Nerve.—The microscopic examination of the path of infection, which is the only way in which real progress can be made in the surgical treatment, have been carried on by Turner and Reynolds [8]

8. Turner, A. L., and Reynolds, F. Esmond: Nasal Mucous Polypi: Intranasal Operation on the Ethmoidal Air Cells; Purulent Leptomeningitis; Death; Autopsy, J. Laryng. & Otol. **42**:525 (Aug.) 1927.

and reported in two recent papers. The first dealt with a case of acute purulent leptomeningitis, supervening on an operation on the ethmoidal air cavities, in which microscopic sections demonstrated that the infection spread along the "lymph" sheaths of the olfactory nerves. On one side, one of the dilated sheaths of an olfactory nerve filament contained numerous polymorphonuclear leukocytes. This infected nerve pathway ran upward and backward through a considerable number of serial sections before piercing the dura mater. At places in the lower part of this "lymphatic" tract the polymorphonuclear leukocytes formed dense aggregations resembling abscesses. Higher up, but at a considerable distance from its entrance into the pia-arachnoid spaces, the whole tract was filled with pus. This was continuous with the pus in the pia-arachnoid meshes.

Turner and Reynolds are of the opinion that the dilatation of the perineural lymphatic sheaths in this case was active, not passive; that the dilated perineural sheath was a reaction to toxins and is comparable to the dilatation of the lymph channels which occurs in association with inflammatory foci in the body generally. They believe that the toxins in this case were a combination of bacteriotoxins elaborated by the microorganisms in the nose and ethmoidal air cells and of cytotoxins derived from autolysis of damaged and necrotic tissue the result of the operation and the subsequent infection of the damaged tissue by bacteria. As there were but few bacteria in the nerve sheath itself, they think it probable that the few micrococci which were present gained entrance to the lymphatic sheaths immediately prior to death and subsequent to the dilation, the invading micrococci being so few in number that they were ingested by the endothelial cells of the lymph sheaths. They believe that had the patient lived longer and the infections continued, the dilated sheaths would have contained pus.

Turner and Reynolds still speak of the "lymph channels," which to me is a mistake as it is somewhat misleading. For while both the pia-arachnoid and the lymphatics are derived from the mesenchyme, and the arachnoid prolongations along the olfactory nerves have a communication in the mucous membrane of the nose with the lymphatic system, the cerebrospinal pathways are a specific system and in no sense should be regarded as lymph spaces, and while it is true that the subarachnoid spaces and their perivascular prolongations within the brain probably have a function somewhat similar to the lymphatics, they contain no lymph. Developmentally, there is no evidence that the lymphatics extend into the central nervous system (Weed and Sabine).

(b) By Way of Haversian Canals with Necrosis of Sphenoid.—In a later case, the sixth in their most valuable study of the paths of infection to the brain, meninges and venous blood sinuses from neighboring

peripheral foci of inflammation, Turner and Reynolds,[9] under the title "A Case of Meningococcal Leptomeningitis," state that their microscopic sections showed that the early acute inflammatory changes in the mucous membrane of the sphenoidal sinus were due to a lighting up of a preexisting chronic inflammation; the acute changes were caused by a streptococcus, and the chronic changes, in all probability, were caused by the same micro-organism. The acute changes in the air sinus were later in onset than the meningitis, and it would seem probable that the meningeal inflammation had some ill defined etiologic relationship to the onset of the later, acute changes in the air cavity. They state that it is certain that there was a double infection; while the changes in the sphenoidal sinus mucosa were due to streptococcus, the acute leptomeningitis was caused by *Meningococcus intracellularis.* The meningeal infection entered through the periosteum of the sphenoid. They believe that the acute osteomyelitis of the sphenoid roof was due to the streptococcus and that it passed from the sphenoidal mucous membrane along the Haversian spaces to the diploe of the osseous wall and thence to the cavernous blood sinus. They refer to the work of Lowndes Yates on the condition of the nasal sinuses in cases of epidemic encephalitis, who demonstrated that a number of cases of encephalitis are associated with an increased power of absorption through the nasal mucous membrane or of the posterior nasal sinuses, the increased absorptive power being due to ciliary paralysis and paralysis of mucous secretion. He asserts that the paralyses "are produced either as a result of chronic rhinitis or from a single heavy infection of the nasal mucous membrane, of the type known colloquially as influenza."

(c) By Nonclosure of Fetal Defect with "So-Called Nasal Glioma." —Guthrie and Dott[10] report a case of "So-Called Nasal Glioma"; "Brain Tissue within the Nose." They say that Paul Berger, in 1890, gave a complete account of the pathology of encephalocele, and recognized that certain encephaloceles were not merely miniature brains surrounded by meninges. He stated that sometimes the meningeal envelope and the nerve cells were so altered that it was justifiable to regard the tumor as a neoplasm, for which he proposed the term "encephaloma." He was of the opinion that there existed a series of transitional forms between the encephalocele and the encephaloma, and that, in consequence, the clinical classification was often a matter of difficulty.

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9. Turner, A. L., Reynolds, F. E.: A Case of Meningococcal Leptomeningitis: Death; Autopsy; Inflammatory Changes in the Sphenoidal Air Sinuses and Cavernous Sinus Thrombosis (Streptococcal); A Case Illustrating Double Infection, J. Laryng. & Otol. **43**:34 (Jan.) 1928.

10. Guthrie, Douglas, and Dott, Norman: The Occurrence of Brain-Tissue Within the Nose: The So-Called Nasal Glioma, J. Laryng. & Otol. **42**:733 (Nov.) 1927.

Dahmann and Müller (1925), in their study of injuries to the cribriform plate, mention the case of a child who died of meningitis four weeks after snare removal of a nasal polypus. At postmortem examination, there was found a dehiscence in the cribriform plate through which passed the stalk of an intranasal encephalocele, which had been mistaken for a polypus and partly removed.

As a result of the microscopic studies of these two cases, Guthrie and Dott conclude that: 1. The occurrence of brain tissue in the nasal cavity as a development from an embryonic rest of the central nervous organ, is sufficiently common in young children to constitute a definite clinical entity. 2. Apart from such cases of encephalocele, intranasal tumors containing glial tissue are extremely rare. 3. The congenital swelling known as encephalocele, not uncommon in the occipital region, may appear on rare occasions in the frontonasal region, and still more rarely may be entirely intranasal. Those swellings are neither hernias of the brain nor true gliomas. They are formed during early embryonic life, before the primitive elements of the skull appear, by the outward growth of a small bud of tissue from the anterior cerebral vesicle. When at a later stage the membranous skull is laid down, this bud of tissue may prevent the complete separation of the estoderm from the neuro-epithelium, and a foramen may remain, through which the encephalocele retains its connection with the brain. In other cases, this connection is not retained, and the bud of nervous tissue becomes entirely cut off from the parent structure.

Surgical procedures on such growths by the nose, of course, is apt to be followed by a suppurative meningitis.

(d) By Venous Connections with the Petrous Apex.—Pietrantoni [11] of the Otolaryngological Clinic and Anatomical Institute of the University of Parma, contributes his work on the microscopic anatomic observations on temporal bones in children by means of injections of Chinese ink. He investigated the relations between the venous circulation of the tympanic cavity and the inferior petrosal sinus, the petrous apex, the sixth pair of nerves and the gasserian ganglion, respectively. Clinically, the most important result was the elucidation of the relationship of middle ear inflammation to thrombophlebitis of the cavernous sinus and Gradenigo's syndrome. He demonstrated that the venous circulation of the tympanic cavity is in direct relation to the inferior petrosal sinus (independently of the internal auditory veins), and that

11. Pietrantoni, L.: Routes of Propagation of Inflammatory Middle Ear Processes to the Apex of the Petrous Bone, the Cavernous Sinus, the Sixth Pair of Nerves and the Gasserian Ganglion: The Venous Route, Arch. ital. di otol. **38**:296, 1927.

there also exist direct venous relations between the tympanic cavity and the apex of the petrous bone, as well as between the latter and the gasserian ganglion and the abducens nerve. He gives a bibliography of twenty-six references, and several illustrations.

(e) By Bony Defect in Tympanic Roof Following Lacunar Absorption the Result of Increased Intracranial Pressure.—Mayer [12] reports a case of death from meningitis on the day following the initial otitis.

> The infection of the meninges had entered by a bony defect, which was the result of increased intracranial pressure on the first day of a right-sided earache with reddening of the tympanic membrane, but no tenderness on pressure at the mastoid process; five hours later he had a paracentesis followed by a serosanguineous exudate. Severe headache supervened almost immediately. On the next day, the patient became confused and died on the same day. Autopsy showed purulent meningitis, especially over the right petrous bone, and the histologic observations revealed a recent inflammation in the tympanum, antrum and cells of the mastoid process with an absence of the bony covering, so that the middle ear mucosa was in contact with the dura mater. The inflammation had passed from within, where it was distinctly marked, outward.

On the basis of the histologic observations, Mayer thinks Wittmaack's view that otogenic meningitis passes into the middle cranial fossa from an arrest of pneumatization and abnormally persistent blood vessels is not sufficient; nor is the assumption of other writers, who consider congenital dehiscences as responsible for the transmission. In the author's case, the bone gaps were not congenital, but of later origin, as shown by signs of bony absorption, for in the petrosquamous fissure, cerebral hernias were found, not from disturbances of bone formation but as the results of increased intracranial pressure. Erdheim contends that the internal cranial pressure may be increased by migraine, nephritis and infectious diseases, while Bartel found hypertrophy of the brain in cases of status thymolymphaticus, as the result of otitic complications. Mayer thinks that his case teaches that in the presence of severe headaches persisting after paracentesis, spinal puncture should be done at once, to be followed by mastoidectomy in order to guard against the onset of meningitis.

(f) By Congenital Bony Defect.—Woodhouse [13] reports a fatal case of brain abscess through a defect in the tegmen tympani. The repeated occurrence of epileptiform attacks indicated a cortical lesion. The brain abscess was found at the operation.

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12. Mayer, O.: The Cause of Origin of Meningitis in the First Days of Otitis Media, Wien. med. Wchnschr. **77**:718, 1927.

13. Woodhouse, W. W.: Cerebral Abscess of Otitic Origin (Birmingham and Midland Ear and Throat Hospital), J. Laryng. & Otol. **42**:685, 1927.

III. MENINGITIS

A. MENINGITIS OF OTITIC AND NASAL ORIGIN

(a) Surgical Anatomy of Meningitis.—Nelson [14] reviews surgical anatomy of the subarachnoid spaces adjacent to the nasal sinuses, and concludes that anterior subarachnoid drainage should be possible in meningitis originating from the paranasal cells bordering on to the cranial cavity.

(b) Meningitis Complicating Superior Longitudinal Sinus Thrombosis. Death Apparently from Circulatory Disturbance of the Brain from Retrograde Thrombophlebitis or Infarct with Increase of the Cerebrospinal Fluid.—Lillie [15] reports three cases of thrombosis of the superior longitudinal or straight sinus by extension from the lateral sinus. Two patients died and one recovered. The clinical symptoms in the two fatal cases began with severe headache and signs of general brain pressure, to which were subsequently added, mental confusion, slight blurring of the papillae, and finally a few days before death, generalized or circumscribed convulsive seizures. In one instance in which the cerebrospinal fluid was at first normal one day before death, it showed thirty-two cells and a positive culture. Autopsy observations showed an organized thrombus of the right transverse and straight sinus. The second case presented organized thrombosis of the left transverse and superior sagittal sinus.

Diagnosis of Superior Longitudinal Thrombophlebitis: Doyle [16] reports a case of thrombosis of the superior longitudinal sinus secondary to suppurative otitis media.

> A woman, seven months pregnant, suffered from a left-sided earache and a purulent discharge which ceased, but two months later, immediately after her delivery she had chills and fever with pain over the left side of the head and left side of the upper jaw, with vomiting; later convulsions developed with hemiparesis of the right side and aphasia. Necropsy showed chronic suppurative otitis media, mastoiditis and necrosis of the adjacent bone and a purulent thrombosis of the superior longitudinal sinus with an organized thrombosis of the left lateral and sigmoid sinuses, while a purulent exudate extended over the superior surface of the left occipital pole.

Doyle offers the following suggestions for diagnosis of superior longitudinal sinus obstruction:

1. In the absence of meningitis, the early onset of apathy or stupor with evidence of thrombosis of the transverse sinus secondary to sup-

14. Nelson, R. F.: Meningitis of Nasal Origin: A Study of Surgical Anatomy, Ann. Otol. Rhin. & Laryng. **36**:701 (Sept.) 1927.

15. Lillie, H. I.: Thrombosis of the Superior Longitudinal or Straight Sinus by Extension from the Lateral Sinus: Report of Three Cases, Ann. Otol. Rhin. & Laryng. **36**:324, 1927.

16. Doyle, J. B.: Obstruction of the Longitudinal Sinus, Arch. Neurol. & Psychiat. **18**:374 (Sept.) 1927.

purative otitis media generally indicates infectious thrombosis of the superior longitudinal sinus by retrograde extension, especially if associated with choked disks and convulsions.

2. When the probable presence of tumor or of inflammatory disease can be excluded, jacksonian seizures, showing progression from one foot to the other, or beginning in the foot and gradually involving the homolateral upper extremity, suggest impairment of the circulation of the cerebral veins and probable thrombosis of the superior longitudinal sinus.

3. Abrupt onset of symptoms of increased intracranial pressure, which follow a fluctuating course, should bring to mind the possibility of thrombosis of the superior longitudinal sinus. The absence of the element of progression after a fair length of time, or actual regression of symptoms, is suggestive of thrombosis of the superior longitudinal sinus. If the syndrome is associated with edema of the eyelids and dilatation of the veins of the eyelids and forehead (if fracture of the skull, orbital and periorbital infections and thrombosis of the cavernous sinuses can be excluded), the diagnosis is established.

(c) Deceptive Cases of Otitis and Sinus Suppuration.—Deceptive Character of Mucosus Intracranial Complications: Hlavacek [17] says that usually in the intracranial complications of middle ear inflammations, the etiology can usually be established without difficulty, as the otitis is outspoken. However, when the otogenic character is not so manifest, mistakes may occur. Four illustrative cases are reported, all following infections by *Streptococcus mucosus*. He says that the typical picture of mucosus otitis shows slight initial symptoms, with sometimes no changes of the tympanic membrane, although there may be grave destruction in the mastoid process followed by intracranial complications. The fourth of the reported cases was one of meningitis in which the tympanic membrane presented only slight catarrhal changes, although the streptococcus was found in pure cultures in the cerebrospinal fluid. Autopsy showed sinus thrombosis and an infection of the meninges. In the antrum and mastoid process, *Streptococcus mucosus* was demonstrable by cultural methods. In cases of mucosis infection, the Hungarian author thinks that the diagnosis must be based on roentgen-ray examination of the mastoid process, on the bacteriologic observations and on the history.

Deceptive Character of Meningitis from Sphenoidal Sinus Suppuration: Cases of meningitis from the sphenoid sinus are more common than is usually believed. An illustrative case is reported by Vlasto [18] in a child, aged 11 years, who died five days after admission to the hos-

17. Hlavacek, V.: Cases of Mucosus Otitis with Atypical (Intracranial) Complications, Folia Otolaryng. **27**:344, 1927.

18. Vlasto, M.: Meningitis of Sphenoid Sinus Origin, J. Laryng. & Otol. **42**:746, 1927.

pital with the diagnosis of either an otitic meningitis or a tubercular meningitis. The postmortem examination showed a basal meningitis with a thick layer of pus spreading between the optic chiasma and the pons. Sphenoidal empyema was not suspected during life, and it was not found until the routine opening of the sphenoid at postmortem examination when both sphenoidal cavities were found to be filled with pus. Vlasto [18] thinks that no postmortem examination is complete, in a case of meningitis, which does not include the opening of the sphenoidal sinus cavities, and that in cases in which an otitic cause of the meningitis has been diagnosed, and in which the operative observations are inconclusive, the possibility of a sphenoidal sinus infection should always be considered.

Unsuspected Sphenoiditis the Cause of Insanity: French [19] reports two cases of insanity from nasal sinus disease.

> The first patient stated that she had committed the unpardonable sin of blasphemy and that she heard the devil's voice tempting her to kill herself. Twenty-five years previously she had been treated for mental depression. Transillumination showed antrums and frontal sinuses dark. On puncture, both antrums contained pus, and x-ray examination showed all nasal cavities to be involved. Under general anesthetic, all the cavities, including both sphenoidal sinuses, were opened by the intranasal route. All showed gross pathology, a quantity of necrosed bone being removed from both ethmoidal regions. When last seen, the patient was mentally normal.
>
> In the second case, a woman gave a history of two months' restlessness, depression and marked insomnia accompanied sometimes by confusion, associated with headaches and tenderness of the left frontal sinus and left maxillary antrum. An operation for intranasal drainage of the left frontal, maxillary and sphenoidal sinuses was followed by marked improvement, the patient showing much more self-control and less agitation.

French states that there were nineteen similar cases reported in the literature.

Watson-Williams [20] also reports a case in which "head attacks" were accompanied by distinct loss of memory, inability to concentrate his mind and an intractable temper. His mentality was so profoundly affected that in the periods of depression he was prepared to end his life. This, Williams attributed to a sphenoidal sinusitis, and stated that fluid obtained at operation was clear and was not suspected of infectivity (mucosis) until submitted to bacteriologic examination.

(d) Measles and Its Relation to Meningitis (Case Running an Unusually Slow Course).—Rosen [21] reports a case of meningitis fol-

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19. French, J. Gay: Infection of the Nasal Sinuses in Relation to Insanity, Lancet **2**:13 (July 2) 1927.

20. Watson-Williams, P.: Proceedings of Royal Society of Medicine, Section of Laryngology and Otology, J. Laryng. & Otol. **43**:126 (Feb.) 1928.

21. Rosen, S.: Otitic Meningitis Running an Unusually Prolonged Course, Laryngoscope **37**:437, 1927.

lowing measles otitis. No operation was performed until six weeks after the onset of the first meningitic symptoms, but the child survived for five weeks. The spinal fluid obtained by puncture contained diplococci.

(e) Tuberculous Meningitis with a Complicating Suppurative Otitis or Sinusitis.—Portmann and Despons [22] point out the relatively frequent simultaneous occurrence of tuberculous meningitis in the presence of suppuration of the ear or the accessory nasal sinuses.

The first patient had been previously operated on for adenoids and tonsils, and for some years had suffered from a unilateral nasal suppuration. Severe headache, unilateral exophthalmos and mild symptoms of meningeal irritation called for operative intervention at which there was found slight ethmoid disease with normal frontal sinuses. The patient died of tuberculous meningitis. Pathologic examination of the ethmoid granulations showed no tuberculosis, so that the meningitis, in all probability, was independent of the ethmoid suppuration. However, it is noteworthy that ethmoiditis is relatively frequent in tuberculous patients, so that it is not improbable for meningitis to result therefrom. The second patient was admitted to the hospital on account of headache, vomiting and suspected incipient tuberculous meningitis, but on account of the onset of earache, she was transferred to the Otolaryngological Department where examination showed tenderness on pressure at the corresponding mastoid apex, mild facial paresis and mild Kernig's symptom. The operation disclosed granulations in the antrum; the dura was exposed at the tegmen tympani. There was a fistula of the semicircular canal. The patient recovered.

The diagnosis as to which is the etiologic factor is rendered difficult by the fact that even the examination of the spinal fluid obtained by puncture in early cases often fails to furnish a reliable guide, unless tubercle bacilli can be demonstrated.

(f) Fractures Through the Ear or Accessory Sinuses with Subsequent Meningitis.—Simont [23] thinks that in meningitis of traumatic origin, trephining of the affected portion of the skull should be performed, with opening of the dura and drainage of the inflammatory focus, after removal of all foreign bodies and bone fragments. If the infected focus is in the accessory sinuses or in the orbit, operation is indicated. In meningitis of otogenic origin, wide opening of the mastoid process and exposure of the dura in the middle and posterior cranial fossa is required. He states that the tympanic cavity is to be opened in every case of trauma in the presence of a chronic ear process. The meningeal spaces which are filled with a large quantity of exudate must be relieved by punctures or permanent drainage of the subarachnoid spaces. An important part is played by puncture of cisterns. Puncture

22. Portmann, G., and Despons, J.: Tuberculous Meningitis and Auricular or Sinus Suppurations, Rev. de laryngol. **48**:377, 1927.

23. Simont, D.: Operative Treatment of Purulent Meningitis, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **11**:209, 1927.

of the ventricles is not advocated by Simont on account of the great danger of an infection of the brain substance. For permanent drainage, the cisterna pontis and the cisterna cerebellomedullaris are the most suitable. For opening the cisterna pontis, it suffices to detach the dura and to open the subarachnoid space in the region of the porus acusticus internus. An analogous procedure is adopted in the drainage of the cisterna cerebellomedullaris. Experience has taught that permanent drainage exerts a much more marked favorable influence on the course of meningitis than the customary spinal punctures.

I wish to call attention to necessity for the walling off of the mucous membrane of the sinus from any possible contact with a dural opening in all cases of puncture of the accessory sinuses, as I have pathologically demonstrated that it is by means of the rapid proliferation of the mucous membrane of the nose through a dural opening that fractures of the posterior wall of the frontal sinus and fistulae with subsequent rhinorrhea is established, in either of which conditions, death from a later meningitis is apt to follow secondary infections of the nose. This can only be definitely avoided by the walling off of the nasal cavity from the dural opening at the time of the primary operation. This procedure was advocated and a series of cases reported by me at the meeting of the College of Surgeons at Wilmington, Del., in April, 1928. I would also question the statement of Simont that all patients with fracture of the temporal bone, which is the seat of suppuration, should be operated on at once, as experience teaches that but few such patients develop meningitis, and, consequently, operations are rarely called for. The presence of a rise in temperature or other meningeal signs should furnish the deciding factor.

Infection by Unhealed Fracture of Ethmoid.—Mollison [24] showed a specimen in which a cerebral hernia projected through the roof of the nose. Death from meningitis occurred within a week. In the discussion, Harrison referred to a similar case in which death occurred within forty-eight hours.

B. SEROUS (LOCALIZED) MENINGITIS (ARACHNOIDITIS)

Ferreri [25] says: Serous meningitic cysts may be small or large, single or multiple; their walls are extremely thin, being formed by the visceral layer of the arachnoid and by the pia. These cysts often develop in connection with the subarachnoid cisterna. Inflammatory infectious or toxic agents act on the soft meningitis and may cause adhesions

24. Mollison, W. M.: Specimen of Ethmoid Showing Healed Fracture, J. Laryng. & Otol. **42**:693 (Oct.) 1927.

25. Ferreri, G.: Contribution to the Study of Sacculated Otogenic Serous Meningitis, Atti. d. clin. oto-rino-laringol. d. Univ. di Roma **25**:201, 1927.

between the layers of the arachnoid and the pia, so that the free circulation of the cerebrospinal fluid is obstructed and its secretion (increased by the inflammation of the meninges) becomes the source of cyst formation.

Ferreri[26] reports a case of circumscribed serous meningitis of the left pontocerebellar angle in a man, aged 64, as a sequel of influenza with suppuration in the left middle ear. About three months after the initial otitis, the patient had left-sided facial neuralgia and a left-sided facial paresis. He then developed a right-sided hemiparesis which promptly became a complete hemiplegia with disturbance of the deep mobility. There was also insufficiency of both abducens nerves, more marked on the left. The patient's mastoid was opened with the escape of pus and blood. Death occurred four days later. Autopsy showed advanced basal arteriosclerosis and leptomeningitis. On removal of the brain, a clear fluid escaped from the cranial fossa, evidently from a serous meningeal cyst. The pons was compressed away from the left and below. Its left half was depressed.

Serous Meningitis as the Cause of Papillary Stasis (Exophthalmos).—Richter[27] points out that septic temperature, meningitic symptoms and protrusion of the eyeball or palpebral swelling are considered as convincing signs of thrombosis of the cavernous sinus, but that these ocular phenomena are frequently missing in otogenic thrombosis of the cavernous sinus, while they are always present in rhinogenic thromboses. He emphasizes that palpebral swelling in the course of middle ear inflammation is not necessarily the result of a cavernous sinus thrombosis, as in his case it was only the manifestation of a serous meningitis, as demonstrable by its being rapidly cured from lumbar puncture.

In obliterative thrombosis[28] the Queckenstedt sign is positive when pressure is applied to the unobstructed side, because the increase of venous pressure caused by the stasis is transmitted to the cerebrospinal fluid, but when the jugular vein of the diseased side is obliterated, the rise of pressure in the cerebrospinal fluid does not occur. However, Lemaitre and Aubin, the French authors, in a case of undoubted jugular thrombosis, operated on a man, aged 46, in whom the compression of the jugular vein of the affected side caused a distinct rise of pressure of from 40 to 45 mm. Accordingly, they believe that the Queckenstedt symptom does not necessarily exclude an obturating jugular thrombosis on that side. Again, it may be absent in the case of complete obliterative thrombophlebitis. Two peculiarities may explain the abnormality in the

26. Ferreri, G.: Contribution to the Study of Serous Encapsulated Otogenic Meningitis, Riv. oto-neuro-oftal. **4**:53, 1927.

27. Richter, H.: Temporary Palpebral Oedema in Otogenic Serous Meningitis, München. med. Wchnschr. **74**:1755, 1927.

28. Lemaitre and Aubin: Queckenstedt's Symptom in a Case of Thrombophlebitis with Serous Meningitis, Arch. internat. de laryngol. **33**:974, 1927.

authors' case: (1) the slow development of the thrombophlebitis, which dated back two months and (2) the existence of a serous meningitis, which persisted a long time after the removal of the infectious focus. The papillary stasis paralleled the course of the intracranial tension, the former disappeared at the same time that the latter returned to normal.

Epilepsy in Serous Meningitis (Significance of Conjugate Ocular Deviation).—Epileptic vertigo is not a true vertigo. In the attack the patient turns himself, sometimes repeatedly. In cases of genuine epilepsy, disturbances of the labyrinthine reactions have been described. So-called auricular epilepsy is interpreted as reflex epilepsy, but several cases in Roger and Reboul-Lachaux's [29] experience indicate a circumscribed meningitis due to the otitic disease in which they noted a conjugate deviation of the head and eye at the beginning of the attack. In a case of epilepsy in the presence of old ear suppuration, the rotatory attacks were suggestive of labyrinthine irritation. The epileptic attack began with conjugate deviation. There were normal labyrinth observations with a hypo-irritability on the side of the old otitis. True epileptic attacks were also present.

Purulent Subdural Meningitis (Pia-Arachnoid Abscess: Abscess in Subdural Space).—Bremond and Pourtal [30] report a case of recurrent cholesteatoma suppuration, in which operation showed a sinus thrombosis; the dura over the cerebellum was discolored and there was a sterile subdural collection of pus. The cerebellum itself contained no pus, but its substance was greatly softened. Cerebellar symptoms did not appear until the sixth day after the operation. Death occurred on the twenty-fifth day.

Hart [31] reports a case of circumscribed purulent leptomeningitis. The abscess was subdural, but the cortex of the brain was involved. The intradural suppurative abscess appeared four months after antrotomy. Recovery followed, and Hart thinks it was favored by the fact that the infection was of four months' duration before the intracranial complication occurred, so that the patient must have acquired considerable immunity. Cultures of the pus gave streptococcus and staphylococcus.

Meningitis of the Bulbar Cysterna.—Symonds [32] describes cases of "cranial nerve palsies" in otitis, on the basis of the "Gradenigo syn-

29. Roger, H., Reboul-Lachaux, J.: Epilepsy with Rotatory Attacks and Old Otitic Lesions, Rev. d' oto-neuro-ocul. **5**:25, 1927.

30. Bremond, M., and Pourtal, J.: Thrombophlebitis of the Sigmoid Sinus and Cerebellar Abscess Through Recurrent Old Middle Ear Suppuration. Aseptic Meningitis; Death on Twenty-Fifth Day, Rev. d' oto-neuro-ophth. **5**:663, 1927.

31. Hart, V. K.: Otitic Meningitis and Brain Abscess: Recovery with Operation, Laryngoscope **37**:208, 1927.

32. Symonds, C. P.: Cranial Nerve Palsies in Otitis Media: The Syndrome of the Posterior Fossa, J. Laryng. & Otol. **42**:656, 1927.

drome" (abducens paralysis and headache in otitis media), which is referred by him to an extradural inflammation in the course of the abducens nerve situated between the two layers of the dura. In the first case, the abducens paralysis was associated with vagus and accessory nerve paralysis. The manifestations began about one month after the acute otitis. The course was benign. The second case presented the same picture, but the patient died from bronchopneumonia. Autopsy showed necrosis of the petrous bone extending to the dura, which at this point was diffusely thickened internally. In the third case, the abducens was free, whereas the tenth, eleventh and twelfth cranial nerves were involved. The palsies began very gradually and remained stationary for many years. Finally, in the fourth case, the seventh, ninth, tenth, eleventh and twelfth cranial nerves were affected. The course in this instance was slowly progressive, giving rise to the suspicion of basilar tuberculosus meningitis.

C. TREATMENT OF MENINGITIS

(a) Simply by Removal of Primary Focus of Infection.—Huenekens' and Stoesser's case shows that removal of the primary infection may cure a complicating meningitis even with micro-organisms in the cerebrospinal fluid. Their patient had both pneumococci and hemolytic streptococci. They [33] report a case of streptococcic meningitis of otitic origin.

A child had bilateral otitis media and mastoiditis which was complicated by meningitis; the cerebrospinal fluid contained pneumococci and hemolytic streptococci. Bilateral mastoid operation was followed by recovery after spinal irrigation had been inefficient. The course of the infection from the middle ear to the meninges was not clear.

(b) By Evacuations of Localized Collections of Infected Fluid.—Pugac [34] thinks that by lumbar puncture, it is possible to differentiate serous meningitis from purulent meningitis, the former not infrequently presenting a favorable course. He believes that through the diminution of the intracranial pressure by repeated punctures, the power of resistance of the affected tissues against the infection is favorably modified and considerably increased.

Pugac apparently disregards the importance of evacuating localized collections of infected fluid at a stage prior to this infection of the general cerebrospinal spaces, which in my opinion, is the most important intradural procedure in the surgical treatment of meningitis, as I believe

33. Huenekens, E. J., and Stoesser, A. V.: Streptococcic Meningitis of Otitic Origin, Am. J. Dis. Child. **33**:799 (May) 1927.

34. Pugac, M.: Significance of Lumbar and Suboccipital Puncture for the Diagnosis and Therapy of Otogenic Meningitis, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **11**:162, 1927.

the relief of the general increase of the intracranial pressure, while of assistance, plays but a small part.

Preobrazensky,[35] on the other hand, appreciates the importance of the evacuation of localized collections, and he advocates the opening of the cisterna pontis lateralis as a supplement to labyrinthectomy by an incision behind the internal auditory meatus. Difficulties arise when opening of the labyrinth is to be avoided. The postoperative course differs from that of the customary mastoid operation by the necessity for more frequent changing of dressings after opening the cistern, as the fluid under unobstructed drainage (which is the objective of the operation) rapidly soaks through the dressings. In view of the generally very unfavorable course of otogenic leptomeningitis, beneficial cistern drainage is indicated.

Aurientis' case[36] shows that drainage of the cerebrospinal fluid in itself is not the curative agent in meningitis. It is the drainage of the area of encysted infected fluid, and this before the general arachnoid spaces are invaded.

A girl, aged 13, with acute otitis, had meningitic symptoms appear on the twenty-fourth day. On the next day, following antrotomy, abundant purulent fluid escaped through the auditory meatus, and this discharge continued in undiminished amount until the patient's death seven days later.

(c) Treatment by Intraspinal Medication.—Pomus and Vossnessenskij[37] say that in the therapy of purulent meningitis of otorhinogenic origin, the physician is confronted with several tasks: (1) removal of the primary infectious focus; (2) neutralization of the penetrated organisms and their toxins; (3) relief of pressure by spinal puncture and drainage of the subarachnoid spaces and cisterns at the base of the brain. The operative procedure for the removal of the primary infectious focus must depend on the existing condition, but in all cases the radical removal of all diseased tissue is indicated. They think that medicinal modification of the micro-organism and their toxins must follow an operation. This may be accomplished in a variety of ways, a direct contact with the affected regions being the most efficient. They believe that methenamine is an especially valuable disinfectant,

35. Preobrazensky, B.: Clinical Significance of Opening the Cisterna Pontis Lateralis in Otogenic Leptomeningitis, Russk. Oto-Laringologija **4**:321, 1927; abstr., Zentralbl. f. Hals-, Nasen- u. Ohrenh. **11**:762, 1926.

36. Aurientis: Case of Otogenic Meningitis with Profuse Spontaneous Escape of Cerebrospinal Fluid Through the Auditory Meatus as a Sequel of Mastoid Trephining, Rev. d' oto-neuro-ocul. **5**:38, 1927.

37. Pomus, N., and Vossnessenskij, G.: Treatment of Purulent Meningitis of Otogenic and Rhinogenic Origin, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **11**:208, 1927.

as after its oral administration it is demonstrable in the cerebrospinal fluid within from a half hour to one hour. The dose varies from 1.5 to 8 Gm. daily. These authors also recommend daily intraspinal injections of methenamine solution 50 per cent, and in addition, a colloid silver preparation. Methenamine iodide, an active synergistic compound of methenamine and iodine, they believe to be a very advantageous preparation. In this connection I would respectfully urge extreme caution as my experience teaches that methenamine, when injected into the central tissue itself, causes necrosis, and it is a well known pathologic fact that the cerebrospinal fluid system is very intolerant to any medication.

On the other hand, Fallas and Schellinx [38] report the case of a child, aged 13, with middle ear cholesteatoma and meningitis, who was operated on by incision of the cerebellar dura and exposure of the meninges of an area the size of a 50 cent piece. Lumbar puncture showed 800 cells. The operation was followed by daily injections of a collodial suspension of silver. Lumbar punctures were repeated every other day. The meningitis slowly subsided, the fluid gradually and progressively contained less cells, and after three months, the patient was entirely cured.

(d) Serum Therapy for Meningitis.—Carco [39] reports a case in which surgical operation on the mastoid failed to cause subsidence of the meningitic symptoms, but a cure was obtained through daily lumbar punctures followed by injection of antistreptococcus serum. And again, in a case with pneumococci in the cerebrospinal fluid, rapid improvement followed on five intraspinal injections of antipneumococcus serum. This method is regarded by the author as the best vaccine therapy ranking next in order of efficiency.

D. EXPERIMENTALLY PRODUCED PNEUMOCOCCIC MENINGITIS AND ITS SPECIFIC TREATMENT

Stewart [40] contributes four papers on the treatment of experimental pneumococcal meningitis. After the induction of meningitis in rabbits, he found that intrathecal serum treatment causes rapid agglutination

38. Fallas, A., and Schellinx, A.: Chronic Middle Ear Suppuration: Meningitis; Operation; Recovery, Scalpel **80**:344, 1927.

39. Carco, P.: Importance of Specific Intraspinal Serotherapy in the Treatment of Purulent Meningitis of Otitic Origin, Arch. ital. di otol. **38**:77, 1927; Riv. oto-neuro-oftal. **4**:91, 1927.

40. Stewart, F. W.: Local Specific Therapy of Experimental Pneumococcal Meningitis: I. Experimental Pneumococcal Meningitis in Rabbits, J. Exper. Med. **46**:391-407 (Sept.) 1927; II. The Production, Pathology, and Treatment of Type I Pneumococcal Meningitis in Dogs, ibid. pp. 409-427; III. Incidental Myelitis, Abscess, and Organization of Exudates, ibid. **47**:1-7 (Jan.) 1928; Local Specific Treatment of Experimental Pneumococcus Meningitis, J. A. M. A. **89**:1316 (Oct. 15) 1927.

and phagocytosis of pneumococci, but this very rarely resulted in a cure. Essentially no phagocytosis occurs in the absence of immune serum. In type 1 pneumococcal meningitis in a dog, after lavage from the lumbar region to the cistern of 50 cc. warm saline followed by 0.2 per cent ethylhydrocupreine hydrochloride, respiration ceased; the pulse continued strong; artificial respiration failed to revive; this was " a drug death." The animal died from the effect of the drug on the respiratory center. The manner of death typifies that regularly seen with overdoses of drug. Respiration suddenly becomes shallow and slow, but the pulse continues strong until some time after breathing has ceased.

He makes the following observations from his experiments:

Abscesses following an experimentally produced meningitis are infrequent; they arise either as direct perivascular extensions of the meningeal disease or at the site of infected purpuric lesions.

In prolonged untreated meningitis of reinfected animals, there is nothing resembling the disappearance of exudate following a crisis as occurs in pneumonia. The tendency is toward organization.

Late lesions are characterized by the marked development of a perivascular clasmatocytic apparatus, similar to, but more extensive than that hitherto reported in experimental herpetic encephalitis.

He advocates quadruple punctures and lavage, for he says experience has shown that no partial sterilization will avail. It must be practically complete; if not, reinfection will almost certainly occur. A single negative cisternal culture suggests sterilization, but the cultures should be repeated daily for at least three days before cure can be assured. But he states that "systematic lavage and treatment with ethylhydrocupreine hydrochloride-serum mixtures by the method of quadruple puncture, have resulted in cures of Type 1 pneumococcal meningitis in dogs."

Cases of Cured Pneumococcic Meningitis.—Globus and Kassanin [41] report a case of pneumococcic meningitis in which the patient recovered after repeated lumbar punctures.

Two weeks previous to admission, the patient had discharging ears without pain for one day. She was then apparently well for ten days. Two days before admission (January 25), she again complained of pain in the left ear. The ear did not discharge. The next day the temperature rose with intense headache and vomiting and the patient became stuporous. Examination showed a temperature ranging between 103 and 106 F.; retraction of the neck; bilateral Kernig sign; pupils reacting to light and in accommodation; no localizing signs and drowsiness. Lumbar puncture revealed a turbid spinal fluid with 12,000 cells with 96 per cent polymorphonuclear. Smear gave gram-positive diplococci. Culture showed a pure growth of pneumococcus, group 4. There were 30,000 white

41. Globus, J. H., and Kassanin, J. I.: Pneumococcic Meningitis, J. A. M. A. **90**:599 (Feb. 25) 1928.

blood cells with 90 per cent polymorphonuclear. Examination of the urine showed a trace of albumin. The ears were apparently normal. For treatment, one intraspinal injection of antipneumococcic serum was given and frequent lumbar punctures were performed. For the first ten days, punctures were made three times a day, each time from 30 to 60 cc. being withdrawn. Repeated cultures showed pneumococcus, type 4. The blood cultures were consistently negative. The temperature ranged from 102 to 105 F. for the first few days with decline soon after each lumbar puncture, followed again by a rise within a short time. On February 11, the temperature rose to 104 F., and the fluid again became thick and purulent.

On the following day, February 12, the lumbar fluid did not flow from the needle. Adhesions had apparently formed at the point of the puncture and gave rise to a block. On February 13, a cisternal puncture was done. Fifty cubic centimeters of yellow turbid fluid was obtained. There were 960 cells with 76 per cent polymorphonuclear. The temperature dropped and the rigidity of neck was diminished. Two days later, on February 15, the fluid was still obtainable by the cisternal puncture, but the number of cells had dropped to 180. The temperature was down and stayed so until February 17, when it again rose. Lumbar puncture was attempted, but fluid was obtained only after air had been injected into the subarachnoid space, when fluid again flowed freely. (Injection of air was employed in the hope of breaking up the adhesion. This resulted in immediate relief.) The patient continued to improve until March 1, when suddenly the temperature rose to 104 F., and the meningeal symptoms reappeared. On the subsequent day, two other cisternal punctures were done with resulting improvement. The temperature remained almost normal. For the first time, the spinal fluid was reported sterile. With continued improvement and freedom from meningeal symptoms, the patient still had an afternoon temperature of 101 F. An attempt was then made to produce inflammatory changes in the meninges by an intravenous injection of *Streptococcus aureus* vaccine. Four injections were administered. The patient improved and was discharged on April 4, at which time she was able to walk around and was almost totally free from meningeal signs, but there was still some tenderness over the lumbar spine. Pain was elicited on testing for the Kernig sign. During the whole time, the fundus did not show any abnormalities; the results of the examination of the ear were negative, and it is noteworthy that in spite of the protracted and often unusually high temperature, the patient did not at any time show toxic or psychic manifestations.

Comments by Reviewer: 1. This apparently was an ear case. The otitis was the first symptom; it disappeared and then recurred immediately prior to the meningitis. 2. The condition was not fulminating, because it never caused any psychic or toxic symptoms, although the patient had a high temperature. 3. The patient was undoubtedly cured by the repeated lumbar punctures combined with the cisternal punctures, the injection of a foreign protein adding materially to the protective reaction.

Harkady[42] also reports a case of pneumococcic meningitis with recovery in which the patient two days after a pelvic operation complained of pain in the right ear, which after twelve hours was followed

42. Harkady, J.: Pneumococcic Meningitis Treated by Serum Therapy with Recovery, J. A. M. A. **90**:597 (Feb. 25) 1925.

by perforation and discharge. Two days later symptoms of meningitis developed; the otologist did not consider the meningitis secondary to the infection of the ear, but believed both the result of infection of the upper respiratory tract. Fluid from the lumbar region showed two or three gram-positive diplococci to a field. Serum was injected in the spine and in the cisterna. They conclude their report as follows:

1. Sterilization of the cerebrospinal fluid after the first injection of Felton's serum.

2. Development of a temporary block in the lumbar region, probably due to adhesions.

3. Reinfection characterized by a rise in temperature and by the recovery of pneumococcus, type 1 from the cisternal fluid.

4. Introduction of serum intracisternally, with prompt sterilization of the cerebrospinal fluid. Cultures from the subsequent specimens of fluid obtained from lumbar puncture did not yield organisms.

5. Negative blood cultures.

6. Serum sickness.

I believe that both of these cases were undoubtedly primarily ear cases. Both of these articles treat pneumococcic meningitis, both type 1 and type 4, as separate entities entirely without references to the degree of virulence. In his opinion it is largely a matter of the virulent activity of the organisms, not entirely of the kind of organisms itself.

IV. BRAIN ABSCESS

A. PATHOLOGY

Hofmann [43] prepared serial sections of brains from twelve cases of otogenic temporal lobe abscess. The cases illustrate the evolution of the temporal lobe abscess, which is dependent on the variety and virulence of the agents and whether it originates from the middle ear or the labyrinth, respectively. The extension of the abscess takes place in the white substance and is restricted to it. This is due to the vascular anastomosis of the gray cortex, while the white substance contains terminal arteries. The suppurative encephalitis extends upward and inward with a distinct tendency toward the lateral ventricle. This the abscess usually involves, thereby inducing a fatal meningitis. The formation of an abscess capsule depends on the rapidity of development of the abscess and its duration. The surrounding brain mass presents inflammatory changes, in part foci of softening.

Johnson [44] reports four cases of brain abscess with excellent illustrations of the gross specimens. All of the patients died without operative evacuation.

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43. Hofmann, L.: Contribution to the Study of Otogenic Temporal Lobe Abscesses, Ann. d. mal. de l'oreille du larynx. **46**:980, 1927.

44. Johnson, G. S.: Brain Abscess, Colorado Med. **24**:229 (July) 1927.

Uffenorde [45] and Dandy [46] both report cases of pneumocephalus of bacterial origin occurring in brain abscesses of otitic origin. Both of their patients recovered. Dandy calls attention to the fact that the gas bacillus of Welch, which causes such havoc when attacking other tissues of the body, is apparently somewhat innocuous in the brain substance, as the patients whom he observed were all cured by continuous drainage.

Association of Fracture of Skull and Brain Abscess.—Caldera [47] says that in the absence of symptoms of brain abscess it is often difficult to decide if the abscess is referable to an old otitis or to a recently incurred traumatism. This decision may be of importance from the standpoint of accident insurance.

He reports the case of a woodcutter, aged 42, who, after right-sided ear suppuration, suffered for years from mild headache. Later, he was struck on the nape of the neck by a falling tree. After a brief period of unconsciousness, he developed severe occipital pain, with clear consciousness for nine days, when he again became confused. The occipital protuberance was tender on pressure. The pulse rate was 56; later it was 48. Lumbar puncture yielded fluid under pressure, but otherwise it was normal. He improved for three days, but then died. The autopsy showed a double fracture, the line of fracture passing from the posterior cranial fossa to the right occipitotemporal suture and across to the left posterior cranial fossa, comprising the left lateral sinus, which was thrombotic. Caldera thinks that the thrombosis which blocked the lumen of the sinus and the resulting circulatory disturbances which led to edema of the brain must be regarded as the cause of death, which was the result of the accident and not as the sequelae of sepsis. There was also present an encapsulated abscess in the right temporal lobe the size of a pigeon's egg. A drop of pus was in the right antrum, and there was an atrophic cicatrix of the posterior quadrant of the tympanic membrane.

B. SYMTOMS OF INTRACRANIAL INVOLVEMENT ASSOCIATED WITH SINUSITIS AND OTITIS

(a) Sixth Nerve Paralysis.—Sear's [48] reports are based on a material of 210 cases, 190 of which were collected from the literature, while 16 are hitherto unpublished cases of other otologists, and 4 represent personal observations. Of the entire material, 81 per cent were cases of acute suppuration. Sears was struck with the

45. Uffenorde, W.: Ventrikeleinbruch und spontanes Pneumencephalon im Röntgenbild bei einem Fall von otogenem Schläfenlappen abscess mit Ausgang in Heilung, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:567, 1927-1928.

46. Dandy, Walter E.: Pneumocephalus of Bacterial Origin, Arch. Surg. **15**:913 (Dec.) 1927.

47. Caldera, C.: Rare Case of Cranial Fracture in a Patient Suffering from Chronic Otitic Brain Abscess, Boll. d. mal. d. orecchio. d. gola. o. d. naso. **45**:85, 1927.

48. Sears, W. H.: Otogenic Pathology in Paralysis of the Abducens Nerve, Arch. Otolaryng. **5**:462, 1927; A Clinical and Pathological Survey of Otogenic Nerve Paralysis of the Sixth Nerve, Laryngoscope **37**:468, 1927.

frequency of delayed paracentesis or spontaneous perforation of the tympanic membrane; in rare cases, the tympanic membrane was not perforated. Headache was absent in 3 of 161 cases; 10 times, it was mild; 148 times, the headache was very severe. Antineuralgic agents usually proved inefficient. Forty-two cases (20 per cent) terminated in death, including thirty-one deaths from meningitis. In the discussion, Fay pointed out that the headaches are probably not always referable to irritation of the trigeminus, but follow on irritation of the sympathetic plexus in the carotid canal or of the gasserian ganglion.

Sixth Nerve Paralysis—A Neuritis Following Influenza: Boonacker and Huizinga [49] observed that the abducens paralyses in mild ear inflammations all occur in the winter months, and always in the course of influenza epidemics. A careful examination of the patients, in the Hospital for Nose, Throat and Ear Diseases of the University of Gröningen, for symptoms of influenza, such as suppurations of the maxillary antrum, showed them to be present in all cases. From this they conclude that abducens paralysis in otitis media is a neuritis. This assumption is supported by the observation that peroneal muscle paralysis occurred simultaneously in one of the patients. The trigeminal neuralgias of Gradenigo's triad are also interpreted as a manifestation of polyneuritis. Meningitic phenomena were not present in any of the cases. Spinal puncture was applied in one case; the cerebrospinal fluid was found to be normal. All the cases followed a mild course. Antrotomy was required in but one case; the transitory abducens paralysis had disappeared before the operation. One case came to autopsy following an intercurrent disease; the apex of the petrous pyramid was normal.

The fact that abducens paralysis occurs only on the side of the affected ear is explained by the authors on the supposition that neuritis usually appears in the vicinity of inflamed organs, for example, the accessory nasal sinuses. These authors have observed abducens paralysis also in middle ear catarrhs, without inflammatory symptoms, which were cured by catheterization, and as these cases also showed trigeminal neuralgias, they are inclined to assume that abducens paralysis in middle ear disease is not by itself an indication for opening the petrous cells.

(*b*) *Diplopia, Probably from Localized Meningitis Associated with Labyrinthitis, Cured by Intratracheal Injection of Arsphenamine.*—Castex,[50] Camauer and Bratto report a case of otogenic sepsis due to acute labyrinthitis, cured by internal surgical therapy.

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49. Boonacker, A. A., and Huizinga, E.: Abducens Paralysis in Acute Otitis Media, Nederl. Tijdschr. v. Geneesk. **71**:2 (July 2) 1927.

50. Castex, M. R.; Camauer, A. F., and Bratto, A.: A Case of Otogenic Sepsis Due to Acute Labyrinthitis, Cured by Internal Surgical Therapy, Prensa méd. argent. **14**:161, 1927.

A man who had suffered for years from a bilateral ear suppuration was attacked by diplopia, followed by vertigo and vomiting. Extirpation of a polyp in the left auditory meatus was followed by chills, a high fever and persistent vertigo. He was operated on, and fungoid masses were found in the semicircular canal. Puncture of the sinus yielded fluid blood, which was bacteriologically sterile. Recovery followed treatment by intratracheal instillation of 0.3 Gm. of arsphenamine.

(*c*) *Aphasia.*—Laignel-Lavastine, Halphen and Djiropoulos [51] report a case of "Wernicke's aphasia in brain abscess of otitic origin."

Case: Symptoms of meningitis, Kernig's sign, vomiting, etc., appeared about two weeks after paracentesis for middle ear suppuration followed by word blindness and word deafness. The diagnosis of abscess in the temporal lobe was made because of the aphasia. Puncture of the temporal lobe yielded from about 3 to 4 tablespoonfuls of thick creamy yellowish pus (*Staphylococcus aureus*) under pressure. The word blindness subsided on the day following the operation, as did also soon afterward the word deafness. Four weeks after the operation, the patient was discharged cured. On reexamination a fortnight later, there was a certain tendency to fatigue and apparently a slight diminution of intelligence.

In the discussion of this case, Baldenweck cautions against the assumption of a complete cure; he had never observed intracerebral abscesses to heal completely, and believes that grave complications, sometimes very belated, are always to be feared in these circumstances.

Localization of the Cortical Center of Hearing—Word Deafness: Watkyns-Thomas [52] discusses the localization of the cortical center for hearing and gives an analysis, with drawings, of the five cases of pure word deafness that have come to autopsy—the cases of Pick; Serieux and Dejerine; Liepmann, and Barrett—and calls attention to the fact that in all of these the lesion is too diffuse to allow accurate localization. He reports a series of cases of injuries of the temple region and discusses the widely accepted view that bilateral lesions of the first temporal convolution produce deafness. He comes to the conclusion that there is no proof of the auditory function of Heschl's gyri, and states that it is possible that the fibers of the auditory tract are not distributed to a singly subscribed area of the cortex, for while the distribution of the optic tract is to the neopallium, it is to be remembered that the olfactory tract, a still younger sense organ than the ear, passes to the rhinencephalon, and that the cochlea is phylogenetically a far younger organ than the eye.

I am of the opinion that this author, like so many others, has confused the cochlea with the labyrinth. For phylogenetically, while this static labyrinth—otolithic system for orientation—is one of the youngest, if

51. Laignel-Lavastine; Halphen, and Djiropoulos: Wernicke's Aphasia in Brain Abscess of Otitic Origin, Rev. d'oto-neuro-ocul. **5**:299, 1927.

52. Watkyns-Thomas, F. W.: Localization of Cortical Center of Hearing, J. Laryng. & Otol. **42**:505 (Aug.) 1927; 593 (Sept.) 1927.

not the youngest, of the sense organs, to which was later added the semicircular canal system—for equilibrium during motion—in the lower vertebrates at about the same time that a primitive cerebellum appeared, the cochlea—the sensory apparatus excitable by vibrations coming from the exterior, viz., hearing—developed first in the amphibians long after the appearance of the simple median or even lateral inverted eye.

It is my opinion that hearing like speech was not evolved by life, solely by the addition of a localized area of cells to the neopallium as is the case with vision, but (as in the case of speech which followed hearing) is an association of function and tracts, although new cortical areas played a part in this association. This view, first propounded by Pierre Marie to explain the pathologic and clinical discrepancies in aphasia, is now generally accepted by physiologists, although I have not heard it applied to hearing.

Schuster and Taterka's [53] case would substantiate this opinion, for they report a case that furnishes some proof that "pure" word deafness may be caused by a subcortical lesion, as in their case the left temporal lesion was entirely subcortical, "the cortex everywhere being intact."

Case: A woman, aged 67, suffered an apoplectic attack with a resulting right-sided hemiparesis. About eighteen months after the onset, she developed loss of understanding for words, noises and melodies which persisted until death two and one-quarter years later. Although her hearing was not affected and she showed absolutely normal hearing tests, with isolated exceptions, she did not understand what was said to her. In repeating words after the examiner, it was found that if she understood the word she repeated it correctly, otherwise she replied wrongly; she could not repeat vowels. Spontaneous speech was good. She wrote spontaneously with both hands and without error. She read without fault, and understood what she read. Her understanding of melodies was disturbed. Simple tunes, either sung or played, were not recognized. She could not sing simple tunes. Noises such as the rattling of keys, handclapping and whistling were not recognized when the eyes were closed.

Necropsy showed a left-sided area of softening which lay along the lateral ventricle, destroying the putamen and internal capsule. The paraventricular softening began in the frontal area and extended backward to the occipital area, forming a small linear focus as it extended backward. The softening in the left temporal area involved Heschl's convolution and the white matter of the first and second temporal convolutions in their posterior thirds. In the right hemisphere, there was a small cyst in the white matter of the anterior central convolution and a small focus of softening in the white matter of the anterior central convolution and a small focus in the right internal capsule. There were also pinpoint foci in the putamen. The medial geniculate body was atrophied on the left.

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53. Schuster, Paul, and Taterka, Hans: The Anatomy and Clinical Aspects of Pure Word Deafness, Ztschr. f. d. ges. Neurol. u. Psychiat. **105**:494 (Nov.) 1926; abstr., Arch. Neurol. & Psychiat. **19**:153 (Jan.) 1928.

The paraventricular focus was unquestionably the oldest; Schuster and Taterka think that it was the cause of the apoplectic attack and had nothing to do with the word deafness since the latter is known to be caused by temporal lesions.

The aphasia, i. e., the word deafness, they think was due to the lesion in Heschl's convolution and to the destruction of the posterior third of the first and second temporal convolutions. The foci in the left temporal lobe not only destroyed its fibers, but also fibers connecting the temporal lobe with the operculum and the frontal lobe. It has therefore been shown that the symptom of pure word deafness can be produced by a focus in the left temporal lobe. Bastian, Dejerine, Pick and others believe that both temporal lobes must be injured, and Dejerine believes that there is diminution in function of both auditory centers. Pick asserted that word deafness followed a bilateral lesion causing central deafness. Strohmayer and Veraguth attributed the word deafness to an incomplete bilateral lesion of the auditory field. Schuster and Taterka offer several hypotheses to explain the word deafness in their case: 1. Deep association fibers between Heschl's and Wernicke's areas may have been destroyed, and in this way it was impossible to bring about a change from unqualified to qualified hearing. 2. This change may have been impossible because the projection fibers to Heschl's convolution may have been destroyed, and hence the normal excitability of Heschl's area was not present.

C. DIFFERENTIAL DIAGNOSIS BETWEEN BRAIN ABSCESS AND ENCEPHALITIS

Cathala and Odinet [54] report on "acute otogenic encephalitis in young children."

A child, aged 10 months, was attacked by acute otitis with cerebral symptoms. The convulsions, which originally occurred on only one side, were suggestive of brain abscess. Antrotomy was performed, and pus was found in the antrum. Bilateral convulsions appeared, and the child died sixteen days later. The autopsy observations showed acute diffuse encephalitis.

Chronic Brain Association with Positive Widal Reaction.—Ferrotti [55] reports the case of a patient who ten years ago had had suppurative otitis media through the instillation of caustic agents in the right auditory meatus (in order to escape military service). Four years later it recurred, combined with painful attacks in the right fronto-temporo-parietal region, with fever and vomiting. The suppuration and vomit-

54. Cathala and Odinet: Acute Otogenic Encephalitis in Young Children, Bull. Soc. de pédiat. de Paris **25**:201, 1927.

55. Ferrotti, C.: An Interesting Case of Cerebral Abscess of Otitic Origin with an Abnormally Long Course, Arch. ital. di otol. **38**:135, 1927.

ing ceased after the performance of mastoidectomy, but the pain persisted. Six years later, the suppuration again recurred. The radical operation was followed, at the end of four days, by continuous fever with diarrhea and splenic enlargement. There was a positive Widal reaction for typhoid fever (1:200). Improvement occurred after about ten days. At the end of another ten days, examination showed coma, bradycardia, no meningism and no fever. Brain puncture, in different directions, was negative. Death occurred two days later. Autopsy showed an abscess the size of a walnut in the right lower parietal gyrus; this abscess was of long standing according to the thickness of its wall and the character of its contents. In the author's opinion, the symptoms of the last six years were referable to the abscess. No signs of past typhoid fever were present, and the author believes that the suppurative process had changed the blood plasma so as to render the Widal reaction positive.

Symptomatic Diagnosis of Brain Abscess.—Russi [56] contributes a very thoughtful paper on the diagnosis of brain abscess. On the basis of personal observations on six patients from 5 to 23 years of age, he points out that fever is present according to the absorption of toxins and to associated diseases of the meninges. Symptoms of brain pressure (almost invariably headaches, usually deeply situated) is usual, depending on the seat of the abscess, but not invariably so. Vomiting is common. More characteristic symptoms of brain abscess (derived from intracranial disturbances) consist of rigidity at the nape of the neck, respiratory disturbances, especially Cheyne-Stokes breathing, and choked disk. The diagnosis is assisted by word deafness (left sphenotemporal region), jacksonian attacks, hemiplegia, paralysis of cranial nerves through compression at the base, especially oculomotor palsies. The author states that the diagnosis of brain abscess is extremely difficult, and that especially in obscure cases the diagnosis must rest on accurate observation of the most minute symptoms. He draws the following conclusions: 1. The diagnosis of brain abscess of otitic origin is always difficult, often impossible. A probable diagnosis can often be made even when a brain abscess does not exist alone, but is accompanied by other intracranial complications. 2. The pathognomonic signs of disturbances of speech and intelligence are not constant. 3. When distinct signs and especially localization symptoms are absent, special importance is attached to a study of minor symptoms or irregularities combined with fever, headache, pulse rate, disturbances of respiration, sensory disturbances, etc.

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56. Russi, P.: The Diagnosis of Brain Abscess of Otitic Origin, Riforma med. **43**:585, 1927.

Lund [57] contributes three articles based on his experiences with brain abscess during the past fifteen years.

According to Lund, the greatest difficulty in the diagnosis of otogenic brain abscesses consists in the absence of a pathogenic symptom sufficient to differentiate it from other intracranial complications of otogenic origin. Otogenic cerebral abscess usually appears in connection with other cerebral complications, the result being a confusion of the clinical picture. The diagnosis depends on the proper estimation of the relative value of the symptoms, and the time of their onset in relation to each other; to the pathology in the temporal bone and to that of the cerebrospinal fluid. Lesions of different nonotogenic origin may also prove misleading, but most valuable diagnostic aid is furnished by exploratory cerebral puncture through the dura. In Lund's fifty-four cases the diagnosis was not made in one third of them. The abscess was localized at operation in only one half of the cases. During the last five years, however, the diagnosis was made in all but a single instance (a child, aged 3 years, with purulent meningitis) in seventeen cases; the abscess was not found in but two of the remainder. Ten patients with otogenic cerebral abscess recovered. Right-sided abscesses are more common than left-sided abscesses, but this is not due to a more anteriorly situated sigmoid sinus on the right side, as cerebellar abscess was observed with about the same frequency on the right and left side. The streptococcus, which is the most frequent organism in epidural perisinus abscess and sinus thrombosis, is relatively less common in otogenic meningitis than the pneumococcus. Streptococcus in brain abscess apparently plays a relatively small part; where the pneumococcus and colibacillus predominate, the presence of the latter greatly endangers the prognosis. The rare occurrence of coli in acute aural diseases is due to the fact that it only becomes pyogenic with diminished resistance of the tissue. Pneumococcic abscesses have a tendency to the formation of membranes, but infected abscesses are usually devoid of membranes and associated with considerable tissue necrosis, consequently the meningitic phenomena are much less marked than the encephalitic symptoms. Meningitis, with few exceptions, is monobacillary, but the majority of brain abscesses are polybacillary; most frequently, the colibacillus and pneumococcus were found in association. The cerebrospinal fluid was free from bacilli in most of the cases; sometimes microbes were found, including a polybacillary growth. This double infection may be utilized for the differential diagnosis, as a polybacillary cerebrospinal fluid would point to the meningitis being

57. Lund, R.: Diagnosis and Differential Diagnosis of Otogenic Cerebral Abscess, Hospitalstid. **70**:695 (July 28) 1927; 719 (Aug. 14) 1927; 743 (Aug. 11) 1927; Acta oto-laryng. **11**:479, 1927.

secondary and to another intracranial complication being present, generally a brain abscess. The demonstration of the colibacillus would also suggest the presence of a brain abscess. In view of the great variability of the cerebrospinal fluid, reliable information is only obtainable through a series of punctures. In the presence of meningeal symptoms, if the pleocytosis suddenly rises it indicates another intracranial complication, viz., a deep epidural abscess, a subdural abscess or a brain abscess. This sudden alteration in the character of the fluid possesses great diagnostic importance. Clearing of the cerebrospinal fluid during the formation of a brain abscess is common, with persistence or aggravation of the intracranial symptoms. With respect to the differential diagnosis between brain abscess and epidemic encephalitis, the latter disease in its chronic form can hardly be confused with otogenic brain abscess, as abscess constitutes a relatively acute disease, with considerable and progressive pleocytosis in the cerebrospinal fluid. In the acute and subacute form of epidemic encephalitis, on the other hand, confusion is possible especially when the encephalitis is combined with meningeal involvement. Here an optic neuritis and considerable mononuclear pleocytosis, in combination with the patient's somnolence and lethargy, which may simulate the slow cerebration of abscess, may lead to a diagnostic error if middle ear suppuration is present. In one of the author's cases of this kind, in a patient who was admitted in a comatose condition, operative intervention was not undertaken because the cellular increase in the fluid was weak in proportion to the severity of the intracranial symptoms, and because the suppuration was quiescent. Lund thinks that the changes of the ocular fundus have the same cause as the pathologic changes of the spinal fluid, and are the result of a toxic action on the cranial contents, but this does not exclude the effect of circulatory disturbances in the venous system. The degree of the aphasia depends not so much on the site of the abscess, as on the extent of the edema and the encephalitis which surrounds the focus. In one half of the abscesses in the left temporal lobe, aphasia was absent; so that the absence of aphasia in right-handed persons by no means excludes an abscess of the left temporal lobe. Small abscesses with an extensive encephalitic zone may cause more severe aphasic disturbances than large well encapsulated abscesses. The remote symptoms in temporal lobe abscess are to be regarded as the influence of the encephalitis on the internal capsule. Babinski reflex possesses value only when in combination with other symptoms.

I note that neither Ferrotti, Russi or Lund, although all their papers deal with the diagnosis of brain abscess, mention the diagnostic aid that may be furnished by the repeated daily taking of the visual field.

The finding of hemiopic undulations of the field—always unsuspected by the patient—will frequently enable the diagnosis of a temperosphenoidal lobe abscess at an early date.

The routine daily examination of the visual fields in suspected brain abscess was advocated by me in "Brain Abscess" [58] in 1922. Later, further studies of the visual fields in intradural suppuration have enabled me to diagnose edema of the brain of the homolateral side, and thus to differentiate between intracerebral suppuration and suppurative meningitis or sinus thrombosis.

This latter work was described by me at the meeting of the combined sections on ophthalmology and nervous and mental diseases of the American Medical Association at Minneapolis on June 14, 1928.[59]

Mechanical Diagnosis of Brain Abscess.—(*a*) Encephalography: Carpenter [60] reports a case of abscess of the brain localized by encephalography. A boy, aged 12, developed meningitis, osteitis and choked disks during the four months before the examination. No localizing symptoms were present. The trephine air test revealed absence of the anterior portion of the right lateral ventricle. A large abscess was drained. He concludes that encephalography by the lumbar puncture method under ether anesthesia, with the patient in the upright sitting position, practically is devoid of the serious reactions met with when made under local anesthesia, and is "not more dangerous than many other useful procedures such as tonsillectomy and appendectomy, except in patients with well developed brain tumors, with abscess or with communicating type of hydrocephalus."

(*b*) Injections of Sodium Iodide: Rockey [61] uses a 20 per cent sodium iodide aqueous solution; as sodium iodide is very soluble, it is rapidly absorbed and furnishes an excellent contrast medium. If injected without undue pressure in an abscess cavity, it does not cause irritation. This does not refer to the thecal cavity.

In a case of brain abscess of post-traumatic origin in a man, aged 59, with a history of compound fracture of the skull in the left parietal region, sustained at the age of 3 years, who had had repeated surgical interventions, a contrast solution study of the abscess was made one week after drainage by injecting a 20 per cent aqueous solution of sodium iodide through a tube. The injection and subsequent roentgenograms were made with his head turned on the side, so that the fluid would not run out. Care was used to exert no undue pressure. Twelve cubic centimeters entered the cavity. X-ray pictures were made. There were no untoward symptoms resulting from the injections of sodium iodide. Improvement was fairly constant at first. The drainage tube was kept in place from Nov. 13,

58. Eagleton, W. P.: Brain Abscess: Its Surgical Pathology and Operative Treatment, New York, The Macmillan Company, 1922.

59. Eagleton, W. P.: Localizing Value of Ophthalmic Examinations in Suppurative Diseases of the Brain, Tr. Ophthalmological Section, A. M. A., 1928.

60. Carpenter, E. R.: Lumbar Puncture and Trephine Methods, Am. J M. Sc. **173**:333 (March) 1927.

61. Rockey, Eugene W.: Value of Radiographic Contrast Solutions in the Study of Brain Abscess, Ann. Surg. **88**:22-30 (July) 1927.

1926, to Jan. 23, 1927, but gradually shortened during the week preceding the latter date. On the morning of January 22, it was found on the dressings. Irritability and headache occurred on the afternoon of this day, followed by general aggravation. Paralysis had again become complete in the right arm and leg. The drainage tube was reintroduced into the sinus, but no accumulation of pus was found on repeated puncture through a newly made opening in the exposed dura, at a point apparently directly over the upper pinched-off abscess cavity. The patient seemed better for two days and then became unconscious, with Cheyne-Stokes respiration. Again, at a depth of one-quarter inch, an abscess cavity was entered and creamy pus evacuated. Autopsy disclosed a meningitis which had evidently involved the left parietal region for a period of some days and then become generalized.

Rockey says that by iodine solution one is able definitely to know the location, size and shape of the abscess cavity and to study the manner in which healing takes place.

D. FRONTAL LOBE ABSCESS

Imperatori [62] reports five frontal lobe abscesses following frontal sinusitis. All his cases were in patients under 28 years of age. All had fronto-ethmoiditis with either an abscess of the orbit, a chemosis or edema of the upper lid. In three cases, the invasion was by direct extension from the frontal sinus. One was a metastatic infection. Three of the patients had convulsions prior to the evacuation of the abscess; one had a temporary aphasia and a pronounced optic atrophy. All the others had slight eyeground changes. The patient with aphasia and optic atrophy had a decided change in temperament. Two of the patients died shortly after operation; one fourteen months later and two are alive, two years after the operation.

Osteomyelitis of the Frontal Bone: How to Avoid Intracranial Complications.—McKenzie [63] states that the opening of subcutaneous abscesses is sufficient for the control of acute osteomyelitis of the long bones, probably because the blood serum, washing through the drained spaces, overcomes the infection. The spreading osteomyelitis of the frontal region cannot be so simply controlled, for this disease possesses a quality which causes it to advance until either the patient dies of an intracranial complication or the whole of the vault of the cranium is necrosed. One may assume then that in flat bone cases the body defenses are too weak to cope with the invader, possibly because the blood serum is deficient in antibodies. Whatever the reason may be, the practical point is that drainage of itself is insufficient to arrest the

62. Imperatori, Charles J.: Brain Abscess (Frontal Lobe) Complicating Frontal Sinusitis, M. J. & Rec. **126**:401 (Oct. 5) 1927.

63. McKenzie, Dan: Further Observations of Spreading Osteomyelitis of the Skull, J. Laryng. & Otol. **42**:293-308 (May) 1927.

frontal bone osteomyelitis, and consequently that the surgeon must treat it as a malignant entity. He must erect defenses or cut barriers well ahead of its apparent limits.

When osteomyelitis starts in an ethmoid it reaches the nasal bones quickly. The first sign is the appearance of a pale swelling of the soft parts at the inner side of the orbit or over the bridge of the nose.

When the disease takes origin in the antrum, the orbital floor with the inferior margin of the orbit seems to be the favorite starting point. But in one of McKenzie's cases, in which it followed the Caldwell-Luc operation, swelling appeared in the cheek and immediately after in the lower eyelid and in the hard palate, so that the whole of the superior maxilla seemed to be involved. McKenzie reports promising results from the use of colloid silver administered intravenously. In three of the four cases in which he tried it, the improvement in the symptoms was unmistakable. He describes an operation for osteomyelitis of the frontal bone, the first step consisting in cutting a gutter in the bone beyond the area of infiltration after reflecting the skin downward. The bone is then thoroughly removed from this point downward to the point of original infection.

E. CLINICAL IMPORTANCE OF SACCUS ENDOLYMPHATICUS

(*a*) *In Nonsuppurative Lesions—Vertigo.*—Portmann [64] contributes four articles on investigations of the saccus endolymphaticus, results and clinical application and gives a detailed description of the saccus endolymphaticus, its situation and topographic relations. He thinks it is of practical importance that the saccus at an advanced age presents a much smaller circumference than in youthful persons. In order to study the physiology of the saccus, the author experimented on selachians (sharks), in which the saccus lies subcutaneously on the skull, opening through a canal to the outside between the eyes so that it is easily accessible for experimentation. The saccus at the surface was obliterated with the cautery, and the movements of the animal were then observed and roentgenograms taken. It was shown that the animals with a closed saccus presented a grave disturbance of equilibrium, indicating that intactness of the saccus is indispensable for a good function of the labyrinth. Portmann thinks that his anatomic and physiologic investigations demonstrate that the saccus, due to its intradural situation, is constantly exposed to pressure in the subarachnoid space, but especially to the intralabyrinthine pressure. This suggests the interpretation of the pressure elevations in the labyrinth, which in

64. Portmann, G.: Acta oto-laryng. **11**:110, 1927; Vertigo, Arch. Otolaryng. **6**:309 (Oct.) 1927; J. Laryng. & Otol. **42**:809 (Dec.) 1927; Surgical Treatment of Vertigo by Means of Portmann's Operation, Riv. oto-neuro-oftal. **3**:658, 1928.

man are associated with severe symptoms such as vertigo, deafness and tinnitus. He does not know whether the increased pressure is of intralabyrinthine origin or is caused by an intracranial rise of pressure. The latter he would regard as analogous to the pressure elevations in the eye ball in glaucoma, for which he coins the term "auricular glaucoma." For its cure he performs a decompression by opening the saccus endolymphaticus (Portmann's operation), which consists of exposure and incision of the saccus. It has been performed by the author with good results.

(*b*) *Suppuration at the Site of the Saccus Endolymphaticus.*—Horne [65] reports observations made at two autopsies. The sinus was involved in both instances; both patients died of lateral sinus thrombosis and meningitis. The two specimens demonstrate the development of a circumscribed abscess at the site of the saccus endolymphaticus. In the first case, the infection had spread from the middle ear to the labyrinth, via the oval window; at autopsy, by stripping the dura mater from above downward to the vestibular quadrant, it could be seen that pus had emerged along the aqueduct to the site of the saccus endolymphaticus. In the second specimen, on stripping the dura mater farther downward to the level of the exit of the vestibular aqueduct, it became evident that pus had tracked within the cordlike prolongation of dura mater, which enters a fissure in the bone at the site of the orifice of the aqueduct and the saccus endolymphaticus. The abscess sac was formed between the layers of the dura mater, and not between the dura mater and bone.

To show the variety of this condition, Fraser [66] stated in the discussion that he had microscopically examined over 100 cases of intracranial complications in 35 of which the labyrinth was involved, but he had found an empyema of the saccus endolymphaticus in but one instance.

F. CEREBELLAR ABSCESS WITH SINUS THROMBOSIS

Missorici [67] reports three cases of simultaneous brain abscess and sinus thrombosis, and emphasizes the rarity of this combination. In all three cases, the abscess caused no symptoms and was first discovered at the time of the operation. All were youthful patients, between 11 and 20 years of age; all died. In the first case, the sinus wall was

65. Horne, J.: The Formation of a Circumscribed Intradural Abscess at the site of the Saccus Endolymphaticus, J. Laryng. & Otol. **42**:818, 1927.

66. Fraser, J. S.: Proc. Royal Soc. Med., Section of Otolaryngology, J. Laryng. & Otol. **42**:839 (Dec.) 1927.

67. Missorici, A. P.: Three Rare Cases of Thrombosis of the Lateral Sinus with Brain Abscesses, Arch. ital. di otol. **38**:335, 1927.

infected, thus giving rise to the abscess; in the second case, the pus spread from the thrombus to the cerebellum by the hematogenous route; in the third case, an extradural abscess was primarily present.

In my opinion, one of the chief diagnostic factors between temporosphenoidal and cerebellar abscess is the presence of a sinus thrombosis. It is my practice to regard every case of sinus thrombosis in which headache develops as potentially a case of cerebellar abscess, for it is my experience, in opposition to the view expressed by Missorici, that the combination of sinus thrombosis and cerebellar abscess is rather common.

Crane, Taylor and Patie [68] express the opinion that thrombosis is a protective process, which proves successful when the blood clot does not become infected. They report a case of sinus thrombosis without clinical manifestations, and another case in which sinus symptoms completely concealed a cerebellar abscess, the latter being found only at the autopsy.

Enterococcus as a Cause of Cerebellar Abscess.—Manconi [69] reports a case of enterococcus (pneumococcic?) infection of the cerebellum emphasizing the malignancy of this micro-organism which he states was first pointed out by Caliceti. The symptoms recurred after a period of complete health.

A woman, aged 53, late in January, 1926, was attacked by right-sided otitis media following influenza. Paracentesis was performed; the suppuration persisting for three weeks, when a retro-auricular infiltration over the mastoid process supervened. At the time of intervention, about two months later, the bone was found to be destroyed, with pus and granulations in the sinus. A bone fistula of the temporoparieto-occipital suture was enlarged, and granulations were found in the sinus. The patient complained of persistent headache. Lumbar puncture on the next day yielded a turbid fluid with scanty polymorphous cells and gram-positive cocci, some in capsules, some arranged in chains. Cultures on nutrient mediums were prepared from the pus found at the operation, as well as from the lumbar fluid. The bacteriologic diagnosis showed enterococci. Positive pointing reaction on the right side and hyperemic papilla of the right eye were noted. Lumbar puncture a few days later showed a clear fluid under normal pressure. The patient left the hospital in an improved condition. About five weeks later, she returned complaining of headache and vertigo. On looking to the right, there was slight rotatory nystagmus. The syndrome described by Babinski as cerebellar catalepsy was present; the patient, when lying horizontally on her back, for a long time and without fatigue, held the right arm as well as the right leg in a most uncomfortable raised position, the forearm and hand separately flexed, while on the left side on the same test, the extremities were dropped in the normal manner. Positive past pointing, deviation in the right upper extremity and adiadokokinesis

68. Crane, C. G.; Taylor, J. M., and Patie, H. H.: Thrombophlebitis of the Lateral Sinus Complicating Mastoiditis, S. Clin. N. Amer. **7**:823, 1927.

69. Manconi, M.: Lateral Cerebellar Abscess Through Enterococcus Infection, Atti d. clin. oto-rino-laringoiat. d. r. Univ. di Roma **25**:105, 1927.

were noted. In walking, the patient tottered, stamped her right foot, with deviation to the right. A lumbar puncture furnished turbid fluid under pressure. During the night, the patient had convulsions of the jacksonian type, and died. Autopsy was not permitted.

The path of the infection of the cerebellum in the foregoing case passed along the lateral sinus.

Manconi thinks that although the enterococci are not easily recognized without cultures, a suspicion of the presence of this very grave infection is aroused by the demonstration of gram-positive forms with an extracellular capsule, their frequent arrangement in pairs and coffee bean shape.

G. TREATMENT OF BRAIN ABSCESS

Hasty [70] recommends operation in two sessions. He thinks that the brain abscess should be approached, if possible, from an aseptic region, far from the mastoid wound, in order to guard against a renewed infection.

Results of Treatment of Cerebellar and Temporosphenoidal Lobe Abscess.—Beck and Pollak [71] state that the prognosis of cerebellar abscesses is the worst of all the intracranial complications, as the structure of the cerebellum contributes to the distribution of the pus. Although the prognosis of temporal lobe abscesses is better, the results are far from encouraging. The authors' experience is based on forty cases observed since 1919, in the Vienna University Clinic. Of these forty cases, thirty-seven were recognized and operation performed, with twelve recoveries (32.5 per cent).

Schlander's [72] contribution also comes from Vienna. He states that successful results of otologic survey in diffuse meningitis and brain abscess are restricted to these patients who come under operative treatment at an early stage, during the incipiency of the condition as it were. About 21 per cent of the patients with temporal lobe abscess in Neumann's Clinic were cured.

Epilepsy Following Cured Temporal Lobe Abscess.—Pons [73] reports a case in which after an attack of otitis in a young man, a brain abscess formed in the left temporal lobe. Opening of the abscess was followed

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70. Hasty, F. Emerson: Brain Abscess of Otitic Origin, Ann. Otol. Rhinol. & Laryng. **36**:678, 1927.

71. Beck, O., and Pollak, R.: Critical Comments on the Surgery and Pathology of Otogenic Temporal Lobe Abscesses (on the Basis of Statistics of Forty Cases), Monatschr. f. Ohrenh. **61**:413, 1927.

72. Schlander, E.: Intracranial Otogenic Complications, Med. Klin. **23**:1290, 1927.

73. Pons, J.: Epilepsy Following Upon Brain Abscess of Otitic Origin, Rev. de Laryng. **48**:270, 1927.

by temporary improvement, with subsequent return of grave cerebral symptoms. Another abscess was opened, and the wound healed. One month later, general epileptic attacks supervened, which in the course of the next year returned about once a month, then somewhat less frequently. He states that epileptic attacks are rare in otitic brain abscesses as opposed to traumatic brain lesions. It is still an open question if operative excision of the cicatrix is indicated in such cases.

I have seen the development of epilepsy several years after the apparent cure of a brain abscess. This child had had two convulsions during the suppurative process.

In this regard, the experimental work of Penfield [74] is important to otologists, for in two papers he discusses the mechanism of cicatricial contractions in cerebral insults. He says that the whole supportive structure of the brain might be called the vasoglial framework, or better, the vaso-astral framework.

The vessels pass perpendicularly into the brain from every surface, carrying with them a pial sheath. Any closure of a cerebral vessel may produce a fluid filled space. Meningocerebral adhesions always form when the pia mater is injured and cerebral tissue exposed. The mechanism of cicatrization in the brain is as follows:

The vaso-astral framework is caught in the mesh of a contracting connective tissue scar, and is drawn toward the scar, thus exerting traction on and causing distortion of the surrounding brain. The presence of macerated cerebral tissue in a brain wound is the predisposing cause of the appearance of such connective tissue. Any implanted material for filling defects produces a violent foreign body reaction. If the implant, e. g., fat or fascia, is viable it is incorporated in a new adhesion, as connective tissue grows rapidly on both sides. He advises that in order to eliminate cicatricial contraction and provide for the formation of a fluid filled space with a minimum of adhesion, injured cerebral tissue should be cleanly removed—the old scar excised —thus leaving a dead space filled with fluid.

Penfield thinks that a contracting scar from brain injury must eventually destroy a larger portion of cerebral tissue than would be destroyed by the initial removal of an equal portion of brain, also the slow contraction of such a scar, (continuing as it does for years) must produce a constant irritation which may well be the starting point for a nervous discharge resulting in jacksonian epilepsy. He advocates the clean incision of a cicatrix in order to convert a contracting scar into a fluid filled space and thus to relieve the brain from abnormal traction.

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74. Penfield, Wilder: The Mechanism of Cicatricial Contraction in the Brain, Brain **50**:499, 1927; Cerebral Cicatrix: The Reaction of Neuroglia and Microglia to Brain Wounds, Bull. Johns Hopkins Hosp. **41**:278-303 (Nov.) 1927.

V. ANATOMIC AND PATHOLOGIC STUDIES OF THE CENTRAL CONNECTIONS OF THE VESTIBULAR AND AUDITORY MECHANISMS

Schaller [75] continues his studies of the vestibular connections of the brain, having previously reported a case of glioma in the fourth ventricle, with involvement of the triangular vestibular nucleus, in which, on account of hypertonus, there was a constant tendency to fall backward when supported in the sitting position. The head was held rigidly on the neck, but not turned. There was an extreme hypertonicity of the muscles of both extended lower extremities. The upper extremities assumed a position of flexion of the forearms on the arms, with spontaneous tremor. There was marked dysmetria, especially in the left upper extremity.

Functional vestibular tests were: "Ears: Drums intact, hearing evidently good. Caloric: cold in either ear gives a drawing of the eyes to the same side, with a large slow nystagmus to the opposite side. Spontaneous nystagmus to either side on looking to that side. Eyes move unequally. There is a corneal anaesthesia on either side (not total). There seems to be some interference with the vestibular cerebellar tract, but more central than cortical."

In this case, Schaller's microscopic studies led him to believe that the increased muscular tonus and maintained attitudes, as well as tonic fits, might well be explained by a central vestibular stimulus originating in the vestibular nucleus because of invasion and irritation by the invoiving tumor and its pressure on this nucleus. This lesion can possibly explain also, or at least in part, the inability to change trunk posture by reason of the increased muscular tonus.

He [76] now reports a case with serial sections of the brain of a tumor of the right acusticus of slow growth. During life, the patient presented marked cerebellar incoordination, but lacked any indications of optic edema, symptoms of intracranial pressure or facial paralysis. Deafness was complete in the right ear and only a loud voice could be heard in the left. The right labyrinth was inactive, as evidenced by the absence of an induced nystagmus by the caloric test. The left labyrinth was active. A spontaneous nystagmus of horizontal type was constantly found on lateral movement of the eyes to the right.

At autopsy, the compression of the brain stem was not marked, as the tumor extended upward and outward at the expense of the cerebellar

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75. Schaller, W. F.: Glioma in the Fourth Ventricle, with Involvement of the Triangular Vestibular Nucleus: Report of a Case with Symptoms of Dystonia, Dysmetria, Tremor and Tonic Fits, J. Neurol. & Psychopath. **6**:281-290 (Feb.) 1926.

76. Schaller, W. F.: Mechanism of Compression in a Case of Tumor of the Cerebellopontile Angle, Arch. Neurol. & Psychiat. **17**:609 (May) 1927.

hemisphere and the middle cerebellar peduncle. Microscopic sections showed a lack of degeneration in the dentate nucleus, superior peduncle and red nucleus; this suggested that the cause of the cerebellar incoordination must be of other origin, and the cause was found in a degeneration of the middle peduncle and of the restiform body and in the degeneration of the descending vestibular root, both of the latter degenerations being dependent on the unilateral destruction of the vestibular nerve root.

Strong's [77] most thoughtful paper should sound a note of warning against the excessive claims of cerebellar localization made by some otoneurologists. He says that it seems fairly clear that the paleocerebellum receives proprioceptive stimuli from the organs of bodily movement by way of spinocerebellar and probably by certain secondary afferent cranial nerve tracts, and also from that proprioceptive organ of the head—the vestibule. Regarding the latter, there is still uncertainty both as to whether and to what degree the cerebellum receives direct root fibers or secondary tracts, and whether the projection is principally on an internal nucleus (the nucleus fastigii), or directly on the cortex; as well as the exact distribution of vestibular impulses on the cerebellar cortex.

VI. DIAGNOSIS OF BRAIN TUMORS

(*a*) *Vestibular and Auditory Tests.*—Alexander [78] contributes an article on "choked labyrinth in brain tumors," in which he states that in all true cases of "choked ear" microscopic examinations show that the "pars inferior," by which he means the cochlea, the sacculus of the ductus reuniens, the ductus and saccus endolymphaticus, is alone affected; while the "pars superior"—utricle ampullae and semicircular canals—is not involved. But he states that at the present time a clinical diagnosis of a combined disease of the sacculus and cochlea is impossible, as the diagnosis of disturbed function of the sacculus is impossible. In the present state of knowledge, it is only in exceptional cases that one is able to differentiate clinically a disease of the vestibular portion of the cochlea from that of the semicircular canal. There are no means of recognizing a disturbed saccular function. Alexander ignores the recent work of Quick who regards the otolithic system as entirely a static end-organ, while the semicircular canals are kinetic. Quick's theories, if substantiated, will furnish a simple clinical means of differentiating diseases of the two systems.

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77. Strong, Oliver S.: Unsolved Problems Suggested by Cerebellar Connections and Cerebellar Histology, Arch. Neurol. & Psychiat. **19:** (Jan.) 1928.

78. Alexander, Gustav: Choked Labyrinth and Its Importance for Diagnosis and Indications in Brain Tumor, Surg. Gynec. Obst. **46:**361 (March) 1928.

Conservatism in diagnostic ability by vestibular and auditory tests must be the result of reading Keschner and Grossman's [79] valuable report on entire material, collected from the services of Mount Sinai Hospital, from 1920 to 1926, inclusive.

The general analysis is as follows:

Vestibular and Auditory Signs and Symptoms in Fifty-One Cases

		Intra-cerebellar	Cerebello-pontile	Brain Stem	Supra-tentorial
Vestibular	Spontaneous nystagmus	23	13	5	1
	Spontaneous past pointing	9	3	..	1
Subjective	Deafness	3	11	..	..
	Tinnitus	2	7	..	..

A. Intracerebellar Lesions: Spontaneous nystagmus was noted in twenty-three cases. It was in the horizontal plane in every case, and in nine cases, it was also in the vertical plane. The slow component was in as many cases to the side of the lesion as to the opposite side.

Keschner and Grossman could not find any discernible relationship between the degree of increased intracranial pressure and the presence of nystagmus. In their opinion, this sign needs further investigation before any diagnostic evaluation can be given as to its precise significance in cerebellar disease, especially in regard to its usefulness as an aid in indicating the side of the lesion.

Spontaneous Past Pointing. This sign was present in nine cases. Only two of the patients past pointed correctly; in the remaining seven the direction of the spontaneous past pointing was so inconstant as to be without localizing value.

Caloric Examination. The results are recorded in twenty-two cases. In two, the examination was unsatisfactory and inconclusive. The results of the tests were in full accord with the neurologic results in six cases. In two cases, the caloric tests wrongly pointed to a lesion in the lobe opposite to that indicated by the neurologic observations. In four, the vestibular tests were reported as normal, but the neurologic observations pointed definitely to a lesion in a cerebellar lobe, in which it was found at operation. In two cases, the neurologic observations pointed to a lesion in the cerebellopontile angle and the cochlear and vestibular tests confirmed this localization; at operation the lesion was found in each case in a cerebellar lobe and not in the angle. Two

79. Keschner, Moses, and Grossman, Morris: Cerebellar Symptomatology Evaluation on the Basis of Intracerebellar and Extracerebellar Lesions, Arch. Neurol. & Psychiat. **19**:78 (Jan.) 1928.

patients showed clinical evidences of a posterior fossa lesion; the vestibular tests confirmed the diagnosis of this lesion, but did not shed further light as to the localization. In one case, on admission of the patient to the hospital, the signs pointed to a lesion in the left cerebellar lobe; three successive vestibular tests, done within one month, showed "nothing localizable"; a fourth examination, two months later, pointed to a "nonlocalizable posterior fossa lesion"; at operation the lesion was found in the left lobe. In another patient, the neurologic tests pointed to a tumor in the left cerebellar lobe; three successive vestibular tests, done within one month, reported "nothing localizable"; a fourth examination, two months later, pointed to a "nonlocalizable posterior fossa lesion"; at operation, the lesion was found in the left cerebellar lobe; the caloric tests confirmed this localization, but when the tests were repeated two days later, they were reported as indicating a lesion in both cerebellar lobes; at operation the tumor was found in the left lateral lobe. Finally, in one case, the neurologic observations pointed to a lesion in the posterior fossa, "side not localizable"; the otologist localized the lesion correctly.

They state that the vestibular tests have proved important in differentiating labyrinthine from intracranial disease. In some patients with tumors of one or the other cerebellar hemispheres, the reactions are sufficiently characteristic to be regarded as confirmatory evidence in localizing the disease; in other patients with intracerebellar disease, the results are often ambiguous and do not afford any aid in establishing a localization, and, taken by themselves, may even lead to erroneous conclusions.

Cochlea. Nerve deafness was present in five cases; it was unilateral in four and bilateral in one; in the latter the lesion was in the right cerebellar lobe; in three of the former, the cochlear involvement was on the side of the lesion; in the fourth, the lesion was in the vermis. In three cases, the nerve deafness was total; in two of these the vestibular reactions with the clinical picture were such as to lead to exploration of the cerebellopontile angle, and in each case a tumor was found in the cerebellar lobe. Four of the five patients with cochlear involvement showed definite increase in intracranial pressure.

B. Cerebellopontile Angle Cases: Spontaneous Nystagmus. Spontaneous nystagmus was present in every case. In nine, the slow component was to the side of the lesion, and in six of these there was also some nystagmus in the vertical plane; in four of the latter there was also a rotatory element to the nystagmus. Two cases showed the slow component to the side opposite the lesion; in two others, the nystagmus was of the same amplitude in both lateral planes; in one of these there was also nystagmus in the vertical plane. In the patient with the

bilateral lesions, the slow component was to the side of the more massive lesion.

Spontaneous Past Pointing. Spontaneous past pointing was noted in three cases. In only one patient was the past pointing correct; in the other two, the direction of the past pointing was so inconstant that it was impossible to evaluate this sign.

Caloric Tests. Caloric tests were done in twelve cases; in ten, the results of the tests confirmed the clinical diagnosis of cerebellopontile angle neoplasm. It may, perhaps, be of interest that in only one of these cases was the caloric response typical of a cerebellopontile angle tumor, and in another patient it was atypical before operation, but became typical one month later. In one case, the clinical diagnosis was tumor in the left angle, where it was found at operation, and the otologist reported that the tests indicated a lesion "other than an angle tumor." In another case, the caloric tests indicated a lesion in the right cerebellar lobe, and the neurologic observations were those of a tumor in the right angle, where it was found at operation.

In evaluating the results of caloric tests, they do not hesitate to state that these are considerably more valuable and reliable in localizing cerebellopontile angle than intracerebellar lesions.

Nerve Deafness. Nerve deafness was present in every case. It was unilateral and on the side of the lesion in twelve, and bilateral in one. It was total in eleven, and partial in two. In the patient with bilateral tumors, it was total on both sides.

Tinnitus. Tinnitus was a symptom in seven cases; in one it preceded the deafness by twelve years, and in the other six, it appeared simultaneously with the deafness. Deafness was complained of by eleven patients, in one of whom it appeared three and one-half years before the tinnitus. Deafness appears early and is almost invariably present in cerebellopontile angle tumors. Tinnitus and deafness, associated with involvement of the trigeminus and of the facial nerve (peripheral) on the same side, are early and almost constant symptoms in cerebellopontile angle neoplasms.

C. Brain Stem Lesions with Cerebellar Symptoms: Spontaneous Nystagmus. Spontaneous nystagmus was noted in five cases; in four it was present in both horizontal planes, and in two of these it was also vertical and oblique; in the case with the fourth ventricle tumor, spontaneous nystagmus was noted only on looking to the right.

Spontaneous Past Pointing. None of the patients in this group past pointed spontaneously.

Caloric Tests. Caloric tests were performed in four cases. They were unsatisfactory in the patient with the fourth ventricle tumor; in another case, definite conclusions could not be drawn; in the remaining two, the tests corroborated the clinical diagnosis of a stem lesion.

(*b*) *Mechanical Means of Diagnosis of Acoustic Tumors.*—Vastine and Kinney [80] described a method of diagnosis of brain tumors by the examination of displacement of the pineal shadow. They state that acoustic neuromas caused displacement in 22 per cent of the cases. Intracranial hemorrhage was shown to be capable of displacing the pineal gland in two cases. The method may possibly be of advantage in the diagnosis of brain abscess.

VII. TRAUMA OF THE PETROUS PYRAMID

Bompet [81] reports a case of loss of cerebral fluid through ear traumatism.

A child, aged 19 months, had stabbed himself in the left ear with a wire. The hemorrhage was checked by tamponage. On the third day, there was an escape of cerebrospinal fluid from the ear, but it subsided at the end of a fortnight.

ADVANCES IN SURGICAL TECHNIC OF INTRADURAL OPERATIONS

Kroiss, Otto and Dielmann [82] describe a new method of puncture of the cisterna magna. Their "deep method" would appear to be much more difficult, but safer than the ordinary superficial method practiced by Ayres.

Control of Hemorrhage from Venous Sinuses by Suboccipital Puncture.—In a communication from the Experimental Department of the Institute for Surgical Neuropathology in Leningrad, Wischnewski [83] states that in operations on the brain, in dogs, nearly all hemorrhage stops or is very slight, respectively, even with injury to large sinuses, if from 5 to 10 cc. of cerebrospinal fluid is withdrawn by suboccipital puncture. On reinjection of the fluid, the bleeding at once begins again. This control is constant in the presence of an intact dura. It also occurs with the opened dura, when no negative pressure exists in the dural sac. The transference of the experiments to the human subject is under way.

Cushing long ago called attention to the necessity of ventricular puncture in operating on patients with cerebellar tumors to control excessive bleeding, and to the fact that bleeding from the brain itself often stops on the performance of a lumbar puncture.

But I am not aware that its application for the control of hemorrhage from the venous sinuses has previously been noted.

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80. Vastine, Jacob H., and Kinney, Kenneth K.: The Pineal Shadow as an Aid in the Localization of Brain Tumors, Am. J. Roentgenol. **17**:320 (March 3) 1927.

81. Bompet, R.: Loss of Cerebral Fluid Through Ear Traumatism, Rev. de espec. **2**:297, 1927.

82. Kroiss, Otto, and Dielmann, Hans: Ueber die Liquorgewinnung aus der Cisterna cerebellomedullaris durch Punktion, München. med. Wchnschr. **73**:1228 (July) 1926; abstr., J. Laryng. & Otol. **42**:411 (June) 1927.

83. Wischnewski, A. S.: Hemastasis in Skull Trepanation and Sinus Injury, Arch. f. klin. Chir. **146**:544 (June 18) 1927.

Abstracts from Current Literature

From the Proceedings of the German Oto-Laryngological Congress, 1927

Ear

THE MORE DELICATE CONSTRUCTION OF THE BONE OF THE NORMAL HUMAN LABYRINTH CAPSULE. MEYER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:297, 1927.

The author shows by the use of fibrillar stains and polarized light that the enchondral labyrinth capsule is not lamellated, but fine fibered and marrow-boned.

EXPERIMENTAL CHOLESTEATOMA. BERBERICH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:311, 1927.

Cholesteatoma was introduced in rabbits by suppuration of the bulla. Growth of the canal epithelium occurred through the tympanic membrane, whether the perforation was marginal or central. Even without perforation the epithelium might grow through the tympanic membrane in the presence of sufficient irritation. The genesis of cholesteatoma of the middle ear in animals resembles the early stages of experimental cancer in animals.

INVASION OF THE INTERIOR OF THE SKULL BY MALIGNANT GROWTHS OF THE TEMPORAL BONE. KREPUSKA, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:312, 1927.

In 56,986 patients there were twenty-five growths of the external ear, thirteen of the middle ear and two of the internal ear (acusticus tumor). The article is illustrated by photomicrographs, and should be read in the original.

THE GENESIS OF OTOGENIC MENINGITIS, WITH REMARKS ON THE OPERATIVE TECHNIC OF MASTOID. HAYMAN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:319, 1927.

Of 146 cases of otogenic meningitis, thirty-eight followed sinus thrombosis, twenty, abscess of the brain, and twenty-two, both abscess of the brain and sinus thrombosis. Sixty-six cases occurred through other channels. The article is well illustrated by photomicrographs. The author describes in detail a number of cases, in which the infection in well pneumaticized mastoids extended underneath the labyrinth toward the tip of the pyramid, and left a focus of pus deep in, with healing of the outer part. From this deep seated focus, infection of the meninges took place in various ways: (1) through the saccus and labyrinth, (2) to the jugular foramen, then along the nerve sheaths and (3) direct through the posterior surface of the petrosa mesial to the porus acusticus.

In order to detect these deep-lying foci the author begins operation by removing the tip of the mastoid; then its upper angle and the zygomatic petrosal cells, and antrum, to the internal plate; then the deep cells between the posterior wall of the canal and sinus; last, the cells behind the sinus.

The author believes that the so-called early operation is a mistake and that better results are obtained by careful observation and operation at the proper time.

THE INFLUENCE OF BACTERIOLOGIC AND ANATOMIC CONDITIONS ON THE COURSE OF ACUTE SUPPURATIVE OTITIS MEDIA. WIRTH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:350, 1927.

The author classifies as favorable those patients who recovered from this type of otitis media without operation and as unfavorable those on whom operation was necessary.

In 230 cases, *Streptococcus hemolyticus* was recovered in 43.5 per cent; *Streptococcus viridans* in 21 per cent; pneumococcus type 1 in 8 per cent; pneumococcus

type 2 in 3.9 per cent; pneumococcus type 3 *(Streptococcus mucosus)* in 18.2 per cent; pneumococcus type 4 in 8.7 per cent; influenza bacillus in 3 per cent; staphylococcus in 11.2 per cent; mixed bacilli in 2 per cent.

Of the patients in whom *Streptococcus hemolyticus* was found one of three required operation. One in every 2.6 patients in whom *Streptococcus mucosus* was found required an operation. Contrary to the usual idea, there were more complications with streptococcus infections than with mucosus infections. In the other infections the number of operative cases was negligible.

Bulging of the posterior part of the membrana tympani is considered unfavorable. Of 113 such cases operations were performed in 45. Of 106 patients in whom there was no bulging of the posterior part of the membrane tympani, 12 were operated on. The author distinguishes two types in the degree of pneumatization: The most favorable were those with evenly mixed small and medium cells. The less favorable were those with large widespread cells. The unfavorable types were those with irregular distribution and large peripheral and small proximal cells, with one large cell or with various combinations of these.

Cases in which the origin was in the throat were more severe, on the average, than those caused by nasal infections. This occurs with a greater general prostration which is also ascribed to cases of influenza in which mixed infections are encountered.

Investigations with Amplifying Apparatus. Von Eicken, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:354, 1927.

By means of a chain of filters to magnify the high tones of speech and to minimize the low tones, patients with nerve deafness were enabled to hear much better. This is not necessary in deafness of the middle ear.

The Treatment of Otolith Vertigo. Vogel, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:366, 1927.

Owing to the occurrence of vertigo after the use of tobacco which is a paralyzer of nerves of the sympathetic system the author tried a stimulant, mainly epinephrine hydrochloride, with excellent result in most cases. A dose of from 0.3 to 0.5 cc. of 1:1,000 solution was used hypodermically. The good results lasted from three to eight days, but the doses had to be repeated. Several of the patients had been treated by other well known methods without success. He also experimented with ephedrine with good results, using 0.025 to 0.05 from one to three times a day. One experiment with a synthetic substitute was successful. The result was not immediate; it sometimes took twenty-four hours.

Supravital Staining of Leukocytes in Acute Otitis Media. Hesse, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:377, 1927.

The use of colloidal stains in acute otitis media, preferably trypan-blue and congo red, which do not stain normal leukocytes, is advocated by the author. Because the cell wall resists their penetration, the author differentiates the degenerated cells, which do take the stain. By the relative increase in the degenerative forms the author claims to discover the lack of resistance and onset of complications, and even to set operative indications.

Indications and Prognosis of Attico-Antrotomy. Beyer, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:383, 1927.

In chronic attic cases removal of only part of the attic wall, with preservation as far as possible of the drum membrane and ossicles is advocated by the author. Cases in which the patient hears conversation with a fairly good upper tone limit at a distance of 2 to 3 meters, and shows improvement on inflation, have a favorable prognosis for hearing. Those in which there is advanced nerve degeneration do not present a good prognosis.

Lewy, Chicago.

Miscellaneous

CONTRIBUTION TO THE DIAGNOSIS OF SINUS THROMBOSIS. LORENZ, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:364, 1927.

The author uses the tuning fork on the mastoid, combined with jugular compression. Normally the tone dies down to become stronger when pressure is released. If this symptom appears only when the healthy side is compressed, and not when the diseased side is compressed, it has the same significance as the Tobey test. The test cannot be used in unintelligent patients or patients who are very ill. The patient is first familiarized with the gradual decline of the tone of the fork, then with the compression of both sides and then with unilateral compression, first on the well side and next on the side that is affected.

ADVANTAGES AND DANGERS OF THE DIAGNOSTIC PUNCTURE OF THE CISTERNA. KINDLER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **18**:369, 1927.

1. Punctures of the cisterna is valuable in determining an earlier evidence of meningitis than the more distant lumbar puncture.

2. The method is more reliable. There is no sedimentation of cells, as sometimes occurs in the lumbar region in normal fluid. The cells are exclusively small lymphocytes. The presence of large mononuclears indicates an irritation of the meninges. The author uses Gruebler's polychrome methylene blue, 1:10, with fresh spinal fluid mixed in the pipet. One drop in Fuchs-Rosenthal chamber is used, and it is counted after from ten to forty minutes in order to give the stain time to reach its best.

The author said there is no danger if the puncture is carefully made, and avoided in cases of block of the cisterna.

LEWY, Chicago.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLOGY AND LARYNGOLOGY

Regular Meeting, April 18, 1928

JAMES A. BABBITT, M.D., *Chairman*

TWO CASES OF OTITIC MENINGITIS. DR. GEORGE B. WOOD.

A negress, aged 22, came to the dispensary of the hospital complaining of discomfort in the right ear. The canal of this ear was blocked by a large polyp around which was exuding a moderately purulent discharge. The left ear also showed a purulent discharge with destruction of the drum. The polyp was removed by torsion, and the patient was allowed to go home. Two days later, she began to have pain in the right ear and right side of her head. This became more severe, and two days later she was slightly delirious at times. She was admitted to the hospital about midnight the next day. Her general appearance was that of a normally developed, well nourished, young colored woman, excited and delirious. The temperature was 103, the pulse rate 100 and respiration 25. The patient was irrational and delirious, though she responded to external stimuli and became more excited.

The canal of the left ear was filled with purulent material; the tympanic membrane was almost completely destroyed. In the right ear the canal showed a thicker creamy pus with destruction of the tympanic membrane. There was no definite swelling or tenderness over either mastoid. The pupils were equal, regular in outline and responded slowly to light. The nasal fossae were free, without discharge. The tongue was heavily coated, and the mucous membrane of the pharynx was rather pale. The tonsils were normal. Neither motor paralysis of the face nor accessory disturbance was present. There was distinct rigidity of the neck, and on flexion a suggestive Brudzinski sign had developed. The chest was normal, except for slight impairment of the percussion note over the right scapula. The breath sounds were normal, and râles were not present. The pulse rate was rapid but regular, and the heart sounds were normal with the exception of some accentuation of the aortic second. The blood pressure was systolic 135 and diastolic 80. The abdomen was normal. The reflexes of the arm were rather active but equal. There were no motor disturbances. The legs were held partly flexed at the knees and hips, and the patient occasionally exhibited minor convulsive tremors of slight duration. The knee jerks were equal but active. Kernig's sign was positive on both sides; the Babinski sign was doubtful, and there was no ankle clonus.

On lumbar puncture, the spinal fluid was turbid and under increased pressure. There were leukocytes too abundant to be counted, but at least 90 per cent of them were polymorphonuclear. The same day a radical mastoidectomy was performed under general anesthesia. There was complete eburnation of the whole mastoid process with minute scattered areas of necrosis and pus. Above there was a larger area of necrosis, and the dura in the middle cerebral fossa was exposed. The antrum itself was small and was reached only after the posterosuperior walls of the canal had been removed. Both it and the tympanic cavity were filled with purulent debris and granulation tissue. On raising the dura around the margins of the exposure in the middle cerebral fossa, no extradural collection of pus was found, though there was some granulation tissue on the dura where it was exposed. The dura was also exposed over the lateral sinus, but in this region it appeared normal.

Improvement did not follow this operation, and the patient steadily became worse. Signs of meningitis were definite, and the spinal fluid contained many pus cells and pneumococci. About an hour before death, all the muscles of the body relaxed.

Careful microscopic examination of the polyp did not show any evidence of dural prolapse. Postmortem examination of the interior of the skull showed some inflammatory exudate around the region of the auditory nerve, and an extradural collection of pus on the anterior surface of the petrous portion of the temporal bone, with a perforation through the tegmen.

A second case was that of a girl, aged 10, who developed an acute otitis twelve days after being struck on the right ear with a snowball. The condition was treated at home until she developed chills and fever and some stiffness of the neck, when she was admitted to the Howard Hospital.

A spinal puncture at this time gave negative observations. The patient had complained of some pain in the left ear, but this ear had not discharged.

The canal of the left ear was free. The tympanic membrane was slightly retracted, with some congestion of the manubrial plexus. There was no tenderness or swelling over the mastoid. On the right, the canal contained mucopurulent secretion. The drum was congested with a posterior perforation which had been previously enlarged. There was no edema of the canal wall. There was slight swelling and some tenderness over the mastoid antrum and tip. There was no swelling of the neck, though the tonsillar lymph node was somewhat enlarged on the left side. With the exception of some congestion of the pharynx the nose and throat were normal. X-ray examination of the mastoids showed cellular destruction on both sides, though more marked on the right.

The diagnosis was probable lateral sinus thrombosis, but there was some doubt concerning which ear was responsible; also probable pneumonia.

Examination of the eyeground showed swollen nerve heads on both sides with distinct signs of optic neuritis of 3 diopters.

The temperature on admission was 104.4 F. and rose during the night to 106.8 F. The pulse rate varied between 130 and 160; respiration was between 40 and 70. The discharge from the right ear was profuse.

A simple mastoid operation was performed. The periosteum was slightly adherent, and the cortex was almost normal, but after its removal the rest of the mastoid process was found to be practically destroyed, and a large perisinus abscess was present. Bleeding from the skin and from the bone was free and difficult to control. The dura was markedly thickened and congested, and the surface was covered with more or less adherent exudate. After iodinizing the surface of the dura, a large hypodermic needle was pushed into what was supposed to be the sinus, but blood was not obtained. The dura was then incised at this point, and a collapsed sinus without blood or other contents was found. A curet introduced posteriorly for 1½ inches failed to produce bleeding, but some shreds or necrotic material were obtained. The internal jugular vein was then ligated by Dr. A. C. Wood. After ligation, the bulb was curetted, and a small amount of bleeding was obtained. Immediately after the operation the child was given 20 cc. of pneumococcus antibodies intravenously and 25 cc. under the pectoral muscle. Although the child's condition during the operation was not good, she reacted well and the temperature came down to 99.8 F., and the pulse rate fell from 180 to 116. Later that night, however, the temperature rose to 104; the pulse rate was 130 and respiration 50 and the child became restless and irritable, perspiring freely and at times becoming irrational.

Twelve hours later, the patient was given 45 cc. of pneumococci antibody serum intravenously, and a blood transfusion was made an hour or two later. About 350 cc. of citrated blood from the patient's father was used. The child's temperature fell after this to 97.6 F.; the pulse rate was 90 and respiration 40, but the patient was in a generally weak condition. The temperature, however, rose to 104 the next morning.

Two days after operation a lumbar puncture showed moderately turbid fluid under definitely increased pressure with about 357 cells, but no organisms on direct smears, the culture of which, however, showed the presence of pneumococci.

From this time the patient ran the usual course of meningitis with the usual termination. Postmortem examination of the wound gave evidence of a collection of pus and thrombosis in the region of the torcula.

THE RADIOLOGIC SIGNS WHICH INDICATE EXTENSION OF INFECTION FROM THE ETHMOID AND SPHENOID SINUSES TO THE BASE OF THE SKULL. DR. G. E. PFAHLER, Philadelphia.

Inflammatory processes about any of the accessory sinuses are apt to extend into the bony wall and give rise to an osteitis. This osteitis is indicated at first by absorption of the lime salts due to the action of the osteoclasts. Later, there is a reaction, and, owing to the action of the osteoblasts, there is an increased deposit of lime salts making the affected bone more dense than normal and also interfering with the cancellous structure. This reaction in the bone has been recognized in the frontal bones and in the mastoid areas in connection with inflammation of these cavities. It is now being recognized in the walls of the ethmoid cells, the sphenoid sinuses and extending into the base of the skull. This involvement of the base of the skull may extend to the anterior, middle or posterior fossa, but most commonly involves the middle fossa of the skull. It is shown on the side in which there is involvement of the sinus. The intimate connection of this bone with the dura will indicate the importance of giving such involvement considerable thought.

DISCUSSION

DR. HENRY K. PANCOAST: The principle of osteoporosis and sclerosis is not new and is always considered in the interpretation of mastoid films. Too little attention is paid to thickened walls of the sinus. Much thickening of the wall indicates an infection of long standing. I doubt whether the posterior clinoid process can be influenced by disease of the sinus.

DR. WILLIAM WHALEN mentioned one case referred to by Dr. Pfahler, in which the patient developed a fatal meningitis following a right spheno-ethmoid operation.

DR. GEORGE B. WOOD: I do not understand how osteitis extending over the base of the skull could be due to disease of the sinus. Has this conclusion been proved by histologic studies?

DR. PFAHLER: I have not had the opportunity to prove my statements by microscopic study.

DR. B. A. RANDALL: Have the cavernous sinuses been examined in these cases? If a thrombosis of the cavernous sinus had been present, exophthalmos would most probably have been present. When an extradural abscess is present, meningitis and even abscess of the brain may occur without a demonstrable dural opening. I should like to call attention to the slowing of the pulse in cases of abscess of the brain. Many observers fail to record this observation in these cases.

CEREBROSPINAL RHINORRHEA FOLLOWING INTRANASAL SURGERY: COLLECTION OF CASES AND REPORT OF ONE CASE. DR. EDWARD H. CAMPBELL.

The escape of cerebrospinal fluid following operations within the nose is apparently a rare occurrence, as only a few cases have been reported in the literature. The spontaneous escape of the fluid and cerebrospinal rhinorrhea following injuries to the head, however, have been the subject of many articles during the past thirty years.

In 1899, Sir St. Clair Thomson reported a case of his own along with twenty others collected from the literature. He selected these cases as representing

spontaneous escape of cerebrospinal fluid from the nose and excluded those cases which followed injuries or which were probably due to intracranial tumors.

These twenty-one cases included nine in which the discharge was undoubtedly cerebrospinal fluid and twelve in which the discharge was most probably cerebrospinal fluid. In addition, he collected eight other cases that were suggestive of an intracranial origin of the nasal discharge. Seventeen of his twenty-one cases showed cerebral symptoms, and Dr. Thomson considered that they might possibly have been due to internal hydrocephalus having as its basis a serous meningitis. Postmortem examination in four of the cases did not give any evidence to disprove his hypothesis.

In a recent search of the literature, Locke found fourteen fatal cases of spontaneous cerebrospinal rhinorrhea in which necropsy was performed. Two of these reports were incomplete, and the remaining twelve showed internal hydrocephalus. In eight the hydrocephalus was due to a cerebral tumor which caused an obstruction within the ventricle system. Two were of congenital hydrocephalus in which rhinorrhea developed during early adult life after the fontanels and sutures had closed, and two were cases of hydrocephalus of the adult type.

Eleven of these twelve reports showed that there were openings between the floor of the anterior cranial fossa and the nasal cavity. In the twelfth case only a thinning and no opening was found.

In 1926, Johnston made a detailed report of twenty-one cases of all types of cerebrospinal rhinorrhea. These included one of his own and twenty collected from the literature published since 1900.

Since the publication of Johnston's article, nine more cases have appeared. Among these there have been only a few that have been attributed either directly or indirectly to intracranial operations.

Dr. Campbell reported the case of an adult female on whom he performed a right ethmoid operation on account of infection in these sinuses associated with bronchial asthma. While in the operating room after opening of the anterior ethmoid cells, a rather profuse thin bloody discharge was noticed in the throat. This later lost the bloody tinge and was obviously spinal fluid. No symptoms with the exception of a generalized headache which lasted several hours were present. Large amounts of fluid were given by mouth to prevent any negative intracranial pressure and the subsequent sucking in of infected material. The head was maintained level with the body. The discharge stopped entirely in twenty-five hours, and complete recovery followed. The patient has been followed for about a year after operation and reports no symptoms to date.

A Device for Making Simultaneous Bilateral Stereoscopic Roentgenograms of the Mastoid Cells. Demonstration by Dr. Henry K. Pancoast.

Clinical Observations of X-Ray Diagnosis of Maxillary Sinus Disease After the Injection of Iodized Oil and Deductions from the Pathologic Reports from One Hundred and Fifty Radical Operations on the Maxillary Sinus. Dr. Matthew S. Ersner.

The maxillary sinus is one of the great sources of focal infection that is frequently overlooked. It may become infected from below by the teeth or from above by the adjacent sinuses. Pus may drain into the antrum from above, resulting in its acting as a reservoir for infected secretions. In addition, it may be diseased on account of infection arising within the antrum itself.

In the patients examined by Dr. John Kolmer, the following classes of diseased mucosa were found: (1) hypertrophic changes—fibromyxoma (polyp), (2) hypertrophic changes of cystic fibromyxoma, (3) early atrophic changes and (4) hypertrophic and atrophic changes.

When the mucous membrane of the antrum has passed the stage of resolution and has reached the point of myxomatous change, the ordinary intranasal operations are of no avail. The final decision for radical operation was based on:

(1) the failure of the mucous membrane to undergo resolution after the establishment of ventilation and (2) the thickness of the mucous membrane, visualized by iodized oil-40 per cent and confirmed by X-ray showing retention of oil and delayed excretions from the antrums.

The roentgen-ray examination after the injection of iodized oil is of particular value in cases of atrophic conditions of the maxillary sinuses. Ordinary examination of the sinuses in these cases is usually negative. With iodized oil one learns: (1) the size and outline of the sinus cavity; (2) the conditions of the mucous membrane and (3) the ability of the ciliated epithelium to eliminate the iodized oil (the normal sinus eliminates the iodized oil in from twenty-four to forty-eight hours).

Some cases may show a colorless mucin-like fluid in the maxillary sinus, which is easily overlooked when puncture and irrigation are performed. Many of these cases simulate allergic nasal conditions.

I feel that intranasal surgical measures will not check the progress of polyp formation nor eliminate focal infection. In a group of 150 cases, the focal symptoms of 30 patients on whom intranasal operation was performed did not improve. In eight cases, radical operations were performed, but the diseased mucous membrane was not entirely removed. These patients, too, still showed evidence of infection. I believe that in the radical operation all the diseased mucous membrane should be removed.

The technic in establishing infection of the antrum is as follows: 1. Nasal infections outside the antrum are eliminated by shrinkage, suction, packing and posture. 2. Transillumination is of value. A clear antrum indicates overdeveloped, normal, atrophic or cystic conditions; darkness indicates pathologic processes. 3. Puncture and irrigation show the presence or absence of pus. 4. Roentgen-ray studies are of great value both before and after irrigation. 5. The introduction of iodized oil followed by roentgen-ray studies gives information regarding the size of the antrum, its topography, the nature of its mucosa and the adequacy of the cilia. 6. In acute conditions, polymorphonuclear cells predominate in the pus of the maxillary sinus; in chronic condition, the mononuclears.

The symptoms of this condition are varied. The respiratory system is most often affected by persisted cough, bronchial asthma and hoarseness. Bronchiectasis may be the sequel. Asthma is often associated with nasal polypus. My cases showed chronic infection of the maxillary sinuses in 80 per cent of the cases of nasal polyp. The statistics of the 150 cases reported are as follows:

1. One patient with a fistula of canine fossa was 8 years of age; otherwise the ages ranged between 16 and 71. 2. From the study of 150 cases, I found that 80 per cent of them were associated with polyps; 25 per cent of the patients had chronic maxillary antrums acting as a source of focal infection, but the symptoms were not referable to the antrum or the nose. 3. Other nasal operations were: submucous resections, 48; turbinectomies, 54; removal of polyps, 24; ethmoidectomies, 6, and sphenoidectomy, 1. 4. Other intranasal maxillary operations were, canine fossa 8, and intranasal maxillaries, 30. 5. Radical maxillary operations were, bilateral Caldwell-lucs, 18; single Caldwell-lucs, 126, and Denkers, 6. 6. A general anesthetic was given in 38 cases and a local in 112, consisting of 10 cases in which supraperiosteal anesthesia was used and 102 in which block anesthesia was employed. 7. Bacteria found (on a basis of fifty cases) were: pneumococcus, 3; mixed types, 3; including 2 staphylococcus and aureus and 1 nonhemolyticus and streptococcus; *Staphylococcus albus,* 4; *Staphylococcus aureus,* 9; *Streptococcus nonhemolyticus,* 15, and *Streptococcus hemolyticus,* 16.

DISCUSSION

DR. JOHN KOLMER: The pathologic changes in this condition correspond in a broad way to those in acute and chronic infections elsewhere. Many cases showed myxomatous changes of the fibrous tissue of the submucosa. Atrophic

and hypertrophic changes were often seen together. Retention cysts were sometimes present in the submucosa. Few bacteria were present, the most common ones being of the staphylococcus, pneumococcus and streptococcus groups.

Dr. Karl M. Houser showed a series of lantern slides demonstrating the value of iodized oil in the study of sinus conditions, and commented on the operative observations in some of these cases.

Dr. William Ershner: Eighteen per cent of the cases reported were of dental origin. In operation, block anesthesia is the method of choice. In the differentation of dental cysts, iodized oil is of particular value.

Dr. Alexander Randall: The X-ray reports are not always satisfactory and sometimes are misleading. In one of my cases, the X-ray showed a breaking down of the mastoid process. Operation did not verify this observation. Only the most expert interpretation of X-ray observations is of value. Transillumination is of great value and should not be discarded for the X-ray.

Directory of Ophthalmological and Otolaryngological Societies

INTERNATIONAL

FIRST INTERNATIONAL OTO-RHINO-LARYNGOLOGICAL CONGRESS

President: Prof. E. Schmieglow, University of Copenhagen.
Place: Copenhagen, Denmark, Time: July 29-Aug. 1, 1928.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: William V. Mullin, Cleveland Clinic, Cleveland.
Secretary: Dr. Gabriel Tucker, Bronchoscopic Clinic, University Hospital, Philadelphia.
Place: Portland, Ore. Time: July 8-12, 1929.

AMERICAN BRONCHOSCOPIC SOCIETY

President: Dr. C. J. Imperatori, 17 E. 38th St., New York.
Secretary-Treasurer: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. Bryson Delavan, 40 E. 41st St., New York.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Hill Hastings, 1136 W. 6th St., Los Angeles.
Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. J. Gordon Wilson, 104 So. Michigan Ave., Chicago.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Luther C. Peter, Physicians Bldg., Philadelphia.
Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Hotel Statler, St. Louis. Time: Oct. 15-19, 1928.

LOCAL

THE COLORADO OTO-LARYNGOLOGICAL SOCIETY

President: Dr. C. Howard Darrow, Metropolitan Bldg., Denver.
Secretary: Dr. Harold L. Hickey, Imperial Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

SECTION ON OPHTHALMOLOGY AND OTO-LARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA

Chairman: Dr. Walter A. Wells, The Rochambeau, Washington, D. C.
Secretary: Dr. J. N. Greear, Jr., 1740 M St., Washington, D. C.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Arthur J. Fort, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. Leon E. Brawner, 157 Forrest Ave. N. E., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Arthur M. Corwin, 25 E. Washington St., Chicago.
Secretary: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Grill Room, Stevens Restaurant, 16 N. Wabash Ave., Time: 6 p. m., first Monday of each month from October to May.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Thomas Hood, Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time:: 6:30 p. m., second Thursday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. J. Raymond Hume, Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital, New Orleans.
Time: 8 p. m., third Thursday of each month from September to June.

SECTION ON OTOLOGY AND LARYNGOLOGY OF THE BALTIMORE CITY MEDICAL SOCIETY

Chairman: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore.
Secretary: Dr. Waitman F. Zinn, Medical Arts Bldg., Baltimore.
Place: 1211 Cathedral St. Time 8:30 p. m., last Friday of each month.

DETROIT OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Ray Connor, Stroh Bldg., Detroit.
Secretary-Treasurer: Dr. William Fowler, Maccabees Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

THE KANSAS CITY EYE, EAR, NOSE AND THROAT SOCIETY

President: T. S. Blakesley, Lathrop Bldg., Kansas City, Mo.
Secretary: Dr. A. E. Eubank, 636 Argyle Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May; the November, January and March meetings are devoted to clinical work.

NEW ENGLAND OTOLOGICAL AND LARYNGOLOGICAL SOCIETY

President: Dr. Gordon Berry, 19 Elm St., Worcester, Mass.
Secretary-Treasurer: Dr. Calvin B. Faunce, 320 Commonwealth Ave., Boston.

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Henry C. Barkhorn, 45 Johnson Ave., Newark, N. J.
Secretary: Dr. Lee W. Hughes, 1019 Broad St., Newark, N. J.
Place: 91 Lincoln Park, South. Time: 8:45 p. m., second Monday of each month.

THE SECTION ON LARYNGOLOGY AND RHINOLOGY OF THE NEW YORK ACADEMY OF MEDICINE

Secretary: Dr. Arthur Nilsen, 40 E. 41st St., New York.
Time: 8:30 p. m., fourth Wednesday of each month from October to May.

THE OTOLOGICAL SECTION OF THE NEW YORK ACADEMY OF MEDICINE

Chairman: Dr. J. L. Maybaum, 12 E. 86th St., New York.
Secretary: Dr. R. T. Atkins, 4 W. 53d St., New York.
Place: 103d St. and Fifth Ave. Time: Second Friday of each month from October to May.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. J. Ivimey Dowling, 116 Washington Ave., Albany, N. Y.
Secretary-Treasurer: Dr. L. Prescott Brown, 33 Bancker St., Albany, N. Y.

CINCINNATI OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Walter Murphy, 707 Race St., Cincinnati.
President-Elect: Dr. Charles C. Jones, 19 Garfield Pl., Cincinnati.
Secretary-Treasurer: Dr. Harris H. Vail, 24 E. 8th St., Cincinnati.
Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

THE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SECTION OF THE CLEVELAND ACADEMY OF MEDICINE

Chairman: Dr. W. J. Abbott, 8314 Euclid Ave., Cleveland.
Secretary: Dr. A. L. Stotter, 1148 Euclid Ave., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. W. J. Mellinger, Santa Barbara, Calif.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Place: Santa Barbara, Calif. Time: April 18-20, 1928.

PHILADELPHIA LARYNGOLOGICAL SOCIETY

President: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 13th & Spruce Sts., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

THE SECTION ON OTOLOGY AND LARYNGOLOGY OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

Chairman: Dr. James A. Babbit, 1912 Spruce St., Philadelphia.
Clerk: Dr. Curtis C. Eves, 1910 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

THE MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. B. Selingstein, 801 Union and Planters Bank Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

EYE, EAR NOSE AND THROAT SECTION OF THE HOUSTON ACADEMY OF MEDICINE

President: Dr. Ray K. Daily, Medical Arts Bldg., Houston, Texas.
Secretary: Dr. L. W. Raney, Post-Dispatch Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of the mouth from September to June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. H. L. Warwick, Medical Arts Bldg., Fort Worth.
Secretary: Dr. A. E. Jackson, Medical Arts Bldg., Fort Worth.
Place: University Club. Time: 6 p. m., first Friday of each month except August.

THE MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Charles J. Coffey, 221 Grand Ave., Milwaukee.
Secretary-Treasurer: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.